"A STUDY OF CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SNAKE BITE INDUCED ACUTE RENAL FAILURE IN GOVERNMENT VELLORE MEDICAL COLLEGE HOSPITAL, VELLORE"

A DISSERTATION SUBMITTED TO

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the degree of

M.D. GENERAL MEDICINE – BRANCH I

DEPARTMENT OF GENERAL MEDICINE

GOVERNMENT VELLORE MEDICAL COLLEGE AND HOSPITAL

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SNAKE BITE INDUCED ACUTE RENAL FAILURE IN GOVERNMENT VELLORE MEDICAL COLLEGE HOSPITAL, VELLORE"** is the bonafide work done by **Dr. J.CHANDRU,** Post Graduate student (2013 – 2016) in the Department of General Medicine, Government Vellore Medical College and Hospital, Vellore under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R. Medical University, Chennai for M.D., Degree (General Medicine) Branch - I, Examination to be held in April 2016.

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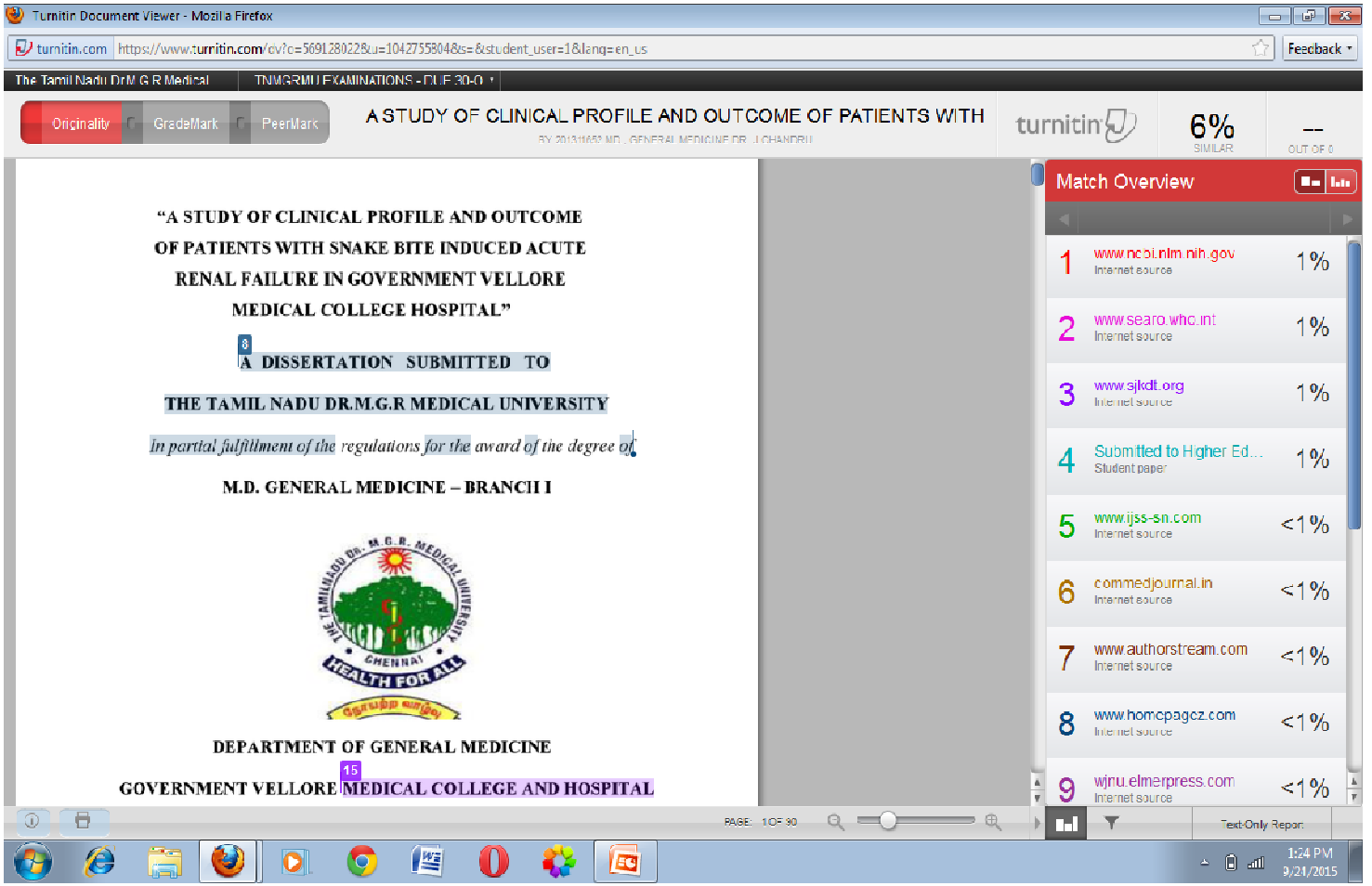
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DECLARATION

I, **DR. J. CHANDRU** solemnly declare that this dissertation titled **"A STUDY OF CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH SNAKE BITE INDUCED ACUTE RENAL FAILURE IN GOVERNMENT VELLORE MEDICAL COLLEGE HOSPITAL, VELLORE"** is a bonafide work done by me in the Department of General Medicine, Government Vellore Medical College and Hospital, Vellore under the guidance and supervision of my unit chief, **Prof.Dr.J.PHILOMENA,Professor & Head of the Department.**

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of M.D., Degree (General Medicine) Branch - I, Examination to be held in April 2016.

Place: Vellore

Date: **DR.J.CHANDRU**

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LIST OF ABBREVIATIONS

ARF	-	Acute Renal Failure
ASV	-	Anti snake venom
ATN	-	Acute Tubular Necrosis
aPTT	-	Activated Partial Thromboplastin Time
BT	-	Bleeding Time
CT	-	Clotting Time
CXR	-	Chest X-ray
DLF	-	Direct Lytic Factor
DIC	-	Disseminated Intravascular Coagulation
ELISA	-	Enzyme Linked Immunosorbant Assay
ECG	-	Electrocardiogram
FDP	-	Fibrinogen Degradation Products
HR	-	Hemorrhagins
PT	-	Prothrombin Time
TT	-	Thrombin Time
USG	-	Ultrasonography
WBCT	-	Whole Blood Clotting Test
WHO	-	World Health Organization

ABSTRACT

BACKGROUND

Snake bite poisoning is known to man since antiquity. It is a well-known occupational hazard amongst farmers, plantation workers, and other outdoor workers and results in much morbidity and mortality throughout the world.

The acute renal failure in snake bite patients largely a preventable complication. So the patients with snake bite should be hospitalized and monitored for early detection of renal complications. This study is an attempt to analyze the clinical profile of snake bite patients and evaluation of acute renal failure in them.

OBJECTIVES

To study the renal involvement in patients with snake bite with reference to clinical features and the time of onset of acute renal failure. To study the course, need for renal replacement therapy including dialysis and outcome.

METHODS

A prospective clinical study of 100 patients of snake bite from August 2014 to July 2015 was done to know the clinical profile and outcome in Government Vellore Medical College Hospital, Vellore and particularly looked for development of acute renal failure. Later each case is studied and investigated with respect to clinical features, course in the hospital, onset of ARF, need for renal replacement therapy including dialysis and mortality due to ARF. Patients were followed till discharge or death. They were treated as per protocol. Clinical data was tabulated. Statistical analysis (SPSS) was done.

RESULTS

The incidence of ARF was (20%), majority of patients were between 31 to 50 years of age. Male patients were predominant with (68%) and (32%) were

female. Most of the patients reported within 4 hours of bite. Common signs and symptoms were pain (99%), swelling (81%), cellulitis (26%), hematuria (26%) and oliguria (20%). Hypotension was present in (15%) of cases which is an important cause of ARF. All the ARF patients showed coagulation abnormalities (WBCT >20 minutes). Hemodialysis was done in 5 patients. Among 100 patients mortality was (2%) and the remaining (98%) recovered completely.

CONCLUSION

Causes of ARF in snakebite was multifactorial in origin. Bleeding and hypotension are among the important causes of ARF. Type of snakebite is another important factor in the development of ARF and Russell's viper bite is more commonly associated with ARF. Lapse of time in presenting to the hospital and abnormal coagulation profile are the predictors of poor outcome. ARF is usually associated with oliguria and generally occurs within 24 hours. Most of the ARF patients had recovered completely with effective management.

KEYWORDS: ARF, Oliguria, Coagulation Profile, Viper bite, Lapse of time.

TABLE OF CONTENTS

INTRODUCTION

SNAKE BITE POISONING is known to man since antiquity. Bite rates are highest in temperate and tropical areas where populations subsist by manual agriculture. In INDIA, a large proportion of snake bites occur when people are working barefoot in the fields or while walking at night. Recent studies estimates somewhere between 1.2 million and 5.5 million snakebites worldwide each year, with 421,000-1,841,000 envenomation and 20,000-94,000 deaths[1].

The principal systemic effects of the envenomation are on the nervous system, kidneys, cardio vascular and blood coagulation and locally at the site of bite. Renal failure complicates 5.5% to 26.0% of all poisoning from snakes. Most cases are due to the Viperidae species of snakes, which includes pit viper, Russell's viper, saw scaled viper, rattlesnake.[2]

The complications related to kidneys are observed in majority of patients with snake bite admitted in the hospital and is an important cause of morbidity and mortality. The onset of renal failure in these patients is signaled by the development of oliguria or anuria. This acute renal failure is largely a preventable complication. So the patients with snake bite should be hospitalized and monitored for early detection of renal complications. Early treatment improves survival in snake bite victims.

This study is an attempt to analyze the clinical profile of snake bite patients and evaluation of outcome in Acute Renal failure in them.

OBJECTIVE OF THE STUDY

1. To study the renal involvement in patients with snake bite with reference to clinical features and the time onset of renal failure.
2. To aid in the prompt diagnosis and effective management of acute renal failure cases in snake bite.

REVIEW OF LITERATURE

HISTORY

Snakes have formed an object of awe and curiosity all over the world. They have been associated with mysticism apart from being objects of fear.[2] Snake bite may be earliest and most common poison known to human being. Myths in the form of stories, dramas and films revolve around dreaded snakes, rivalry of snakes, mating of the snakes. Snakes are worshipped in India and the auspicious days are marked in the name of snakes.[3]

History of snakebites is as old as any civilization and sculptures. The earliest scientifically documented reference to Indian snakes available might be credited to Dr.Patrick Russell. He differentiated venomous and non-venomous snakes especially on viper, Viper Russelli, which is appropriately named after him.[4,5] Sir J. D. Fayrer (1873) carried out detailed study on the physiology of poison of Indian snakes and author a book titled “Thantoophidia of India” in the year of 1874. Col. Frank Walls (1908) strived hard to add our knowledge of the habits and distribution of snakes in his work entitled “The Poisonous Terrestrial Snakes of our British Indian Dominions and How to recognize them”. It was Col. Gharpurey (1935), medical man turned ophiologist, who first attempted to dispel the ignorance and superstition woven around the Indian snakes, both venomous and non-venomous. He wrote book called “Snakes of India” in semi technical knowledge.

Epidemiology

Snakes are present all over the earth surface except some islands, Arctic and Antarctic. Snakes are poikilotherms and are active around 25-35 degree Celsius. These reptiles have venomous apparatus which consist the fang and venom gland.

Identification of snake type is most important because clinical manifestations depends upon species type. Sometimes the fang mark could not visible (Krait). The killed snake brought makes the identification easier. In this case monovalent-ASV may given

Snake Type

The important poisonous snakes belong to three major families.

1. Vipridae family consist two sub families, a. viperianae and crotalinae
2. Elapidae families have Krait, Cobra, Corel snakes and all Australian venoms snakes.
3. Hydrophidae (Sea Snakes)
4. Colubridae and Atractaspididae are two other families in which few are toxic.

Global scenario

Snake bite is a global issue; it is a most common occupational hazard. The victims are farmers, plantation workers and others belongs to out-door activities in the tropical areas. The fishermen, swimmers and divers are attacked by sea snakes.[9] The morbidity and mortality is high in this group.

Exotic and poisonous snakes are becoming popular as pets in the western world. In United States 25% of bites occurs during handling of snakes.[9] In whole world around 3500 species are identifiable. Among these 350 are poisonous.[7]

Indian scenario

In India 216 species are identifiable and 53 are venomous.[8] Till now there is no reliable statistics are available. Annually 200,000 snake bites are encountered among them 15 – 20000 were died.[14]

In west Bengal at Burdhwan district snake bite incident is 0.16%, and mortality rate is 0.016% annually.[10] In Maharashtra incident rate is 76 bites per 100,000 population with mortality rate of 2.5 per one lakh per annum.[11] Tamilnadu, Maharashtra, Kerala, West Bengal, Uttar Pradesh and Karnataka are the states in India with high prevalence rate of snake bites.[12]

Age and Sex

Although snake bite affects all age group, the majority fall between 11-50 years of age. Interstate studies in India showed 72% of victims are fall in this age group. The study of Banerjee et al declared that gender distribution rate of male and female is 3 : 1[15].

Host and environmental factors

Since most of the incidents are happening in remote areas, the detail of statistical analysis of snakebite is difficult and the data availability is incomplete. As an occupational Hazard, it affects herdsmen, farmers, hunters, plantation workers and workers on development sites are mostly affected.[16]

Snakebites are more common in summer and in the rainy season, thus show seasonal variation[7]. Males are affected more than twice than females, and bites are more common in lower extremities in negligible envenomation. The reason for snakebite is provocation and are inflicted when mistakably trodden.[15-18] Sometimes poisonous snake produce dry bites, that results in negligible envenomation varies from 10-80%.[19]

Mortality

The mortality varies 2-11% in various studies.

Characteristics of Snakes

Snakes body is elongated and consists of head, tail and body regions. The body of snake is covered by scales that are imbricate in primitive snakes and form a distinctive covering in other snakes. Special scales are developed on the head and characterizes for identifying different snakes.

Figure 1: Short, permanently erect, fangs of a typical elapid.

The snakes don’t have eyelids and the pupils are vertical. Snakes have one pair of internal ears which is primitive and there is no external appendage’s. The lower jaw in the snakes has a pair of bones connected centrally in front by a firm elastic ligament and it does not articulate with maxilla as in mammals. The snakes have a row of teeth on either side of the premaxilla and mandible. Venomous type snakes have one pair of fangs on premaxilla. The channel of fangs pours the venom. In Cobra the channel is gutter in nature but in R.viper appears as a hypodermic needle. The fang ends in the venomous gland present on both sides of the jaw underneath the orbit. Ventrally this gland extends as a venomous duct which ends in ampulla.

Figure 2a : Monocellate cobra (Naja kaouthia)

Figure 2 b: Detail of hood

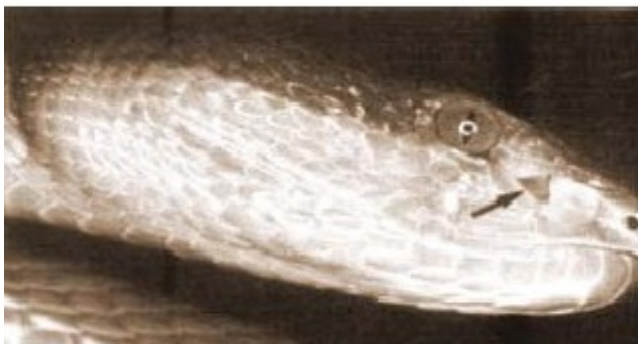


Figure 3: Head of a typical pit viper – white-lipped green pit viper (Trimeresurus albolabris) showing the pit organ situated between the nostril and the eye.

Figure 4: Russell's vipers' details of fangs.

Habitat of Snake[14]

Snakes are carnivorous reptiles and also belong to poikilothermic; it uses venomous apparatus to procure the food.

It has very close range of limited vision and active on movable object with immediate vicinity.

Approach to Identify Snake Types[21]

1. If the tail is laterally compressed, the snake is a sea snake and it is poisonous.
2. If the tail is round, proceed to examine the ventral scales, if the ventral scales are narrow or absent, the snake is non-poisonous. If the ventral scales are broad, extending across the belly, it may or may not be poisonous, to differentiate next examine the head.
3. If the ventral scales are enlarged and only infra labials are present and the last is the largest, it is a krait.
4. If the head is covered with scales and not with shield the snake is a viper and it is poisonous.

Medically important snakes in India[22]

Only six species are responsible for snakebite envenomation in India. They belong to family elapid-cobra and kraits; the family viperidae includes Russell's viper and saw scaled viper .The family hydrophidae includes several types of sea snakes.

Elapids

Elapids consists of King Cobra, Coral Snake, Common Krait, Cobra and Banded Krait

Cobra :(Najanaja)[20]

Cobra seen throughout in India, they are oviparous. It is 1.2 meter length and it is black color, but it varies place to place. The size of the head and neck are same. The pupils round in shape. The fangs are short, grooved and fixed with premaxilla and it lined with mucous membrane. Because of this it cannot bite over clothes and delivers less dose.

Fig 5: Showing king cobra

King Cobra [Raj Naga or NajaBungarus]

King Cobra habitat in forest and the near vicinities[2]. It grows up to 5 meters length and bigger than normal cobra. The colour of king cobra is Black/ Yellow / Brown or Green, but young one would be jet-black. You can see the white and yellow colour cross bars on the body. There is no spectacle mark on the hood. Tails scales are proximally completed and distally separated.

Common Krait [Manyar / Bungaruscaerulus]

This snake grows to a length of 1 to 1.25 meters or even 1.50 meters.

Fig 6: Showing common krait

It is widely seen in India and lives in and around near the residences, thus the reason for more number of snakebites[20]. The colours of the common krait is generally glittering black and have single / dual white bows across the top in the frequent distance. You can see strip of hexagonal scales present on the belly as well as on the back. The head has large shields and scales in tails are complete.

Viperidae

It belongs to ovi-viviparous group. The head of a viper is triangle, and the neck is narrow compared to head with laurel shields and it has elliptical pupils. It has long movable canalized fangs, and it look like hypodermic needles.

It has two different sub families as below

a. Pit Viper – Rattlesnake
b. Pit less Viper – Russell's & Saw Scaled

Pit vipers [Crotalinae] detect warm-blooded food through the special sense organ called pit organ which is resides between the eye and nostril.

Common Green Pit Viper [Hara Phisi/Bamboo Snake/Lachesisgramineus]

Fig 7: Showing common green pit viper

This type of snakes normally lives in the mountains and broadly spread across India. You can see this in various sizes ranging from 1 feet to 4 feet. Normally it is in bright green colour, infrequently in brown or yellow. It has triangle shape of head, flat broad body with a pit between the nostrils and eye. Half white line appears on the flanks, with scales appears on the long tails.

Russell's viper: (Daboia, charn viper, Ghonus or khodchitro)

Fig 8: Showing Russell's viper

It is found all over India except in thick forest. It grows up to 5 – 6 feet[20]. It is in brown / beige colour. On the top it has 3 strips of almond designed spots. Compared to other poisonous snakes it is stouter and with bigger nostrils. It gives typical hissing noise while about to bite. One can easily identified by

a. Discrete V mark with its apex pointing forward on the flat triangle head
b. Head has tiny scales.
c. Extensive whole stomach scales.
d. Short thin tail with shields spliced in to 2 rows.

Saw scaled viper [Echis or Afai/Echiscarinata/Phoorsa]

You can see this in brown / greenish brown colour and in different sizes ranging between 2 and 3 feet. This is also seen across India

Fig 9: Showing Sawscaled viper

Way to identify this snake

1. Like other vipers it has triangle shape head, with an arrow mark in white colour.
2. Flank has curvy line, with a shape like diamond appeared between the 2 curvy lines.
3. Tiny scales in the head.
4. Wide stomach scales.
5. Tail shields are not splinted.
6. Body scales look like saw.

This produces a odd swishing noise while it moves, due to uneven scales on the bottom.

Hydrophidae [Sea snake]

This can be found in the coastal localities. It has Small head with swimming capability through its rubbery flat tail. Even though these snakes are poisonous, bites occasionally. Nostrils are there near the nose with valves, and they breathe freely through the same. Narrow belly plates with tuberculated plates on their bottom. Venom kit is soft and located posteriorly with small fixed teeth.

Fig 10: Blue spotted sea snake

Snake venom[23-25]

In our country 52 [25%] types of snakes are poisonous out of identified two hundred types of snakes. The Common Cobra, Common Krait, R.Viper & Saw Scaled Viper are high poisonous.70% of bites are related to Saw-Scaled snakes, 25% are related to Russell's. Insignificant percentage related to Kraits / Cobras. Small snakes bites also to be considered as poisonous snake bite.

Physical properties of venom[14]

Snake venom is faded see through yellow and is viscous in consistency. It is acidic with gravity between 1.03 and 1.07. It look like crystal once it is dried, will melt in water. Exact quantity of venom spit while bite by snake is unaware, but deadly dose as below

S.no	Snakes	Deadly Dose
1	Cobra [Common]	120 mg
2	R.Viper	150 mg
3	Krait [Common]	60 mg
4	Saw Scaled Viper	80 mg

Chemical composition

Venom contains protein (90 To 95 %) and non-protein (5 to10 %) compounds. Ninety Percentage of the dry weight is protein.

Four broad categories of Toxic component of venom area as below[6]

1. Polypeptides
2. Enzymes
3. glycoproteins and
4. Low molecular weight Compounds

Pharmacological properties

Snake venom might have 5 to 15 enzymes, 3 to12 non-enzymatic proteins, peptides and minimum six other substances. The snake venoms therefore exert a collective action on almost all organ system and on every cell. Clinically speaking however, the more important actions are those on the cardiovascular system, blood, neural system, kidneys and lungs[14]. Of the various enzymes present in the venom, certain of them have been shown to be responsible for some of the significant effects of the poison. Polypeptides components of venom is small, so it absorbed rapidly reaches systemic circulation, produce toxicity in many vital organs and pre/postsynaptic terminals .

a. **Proteinases** abundantly present in viper and crotalid venoms, are proteolytic enzymes and cause marked tissue destruction[17].
b. **Hyaluronidase** is present in almost all snake venoms. This component aids spread of toxins in tissue planes which helps proteolytic enzymes to cause blistering, local oedema, and necrosis.[17]
c. **Cholinesterases** are important enzyme constituents of elapid venom and catalyze the hydrolysis of acetyl choline to acetic and choleic acid. It has been claimed to have a curare like effect and thus contribute to neurotoxicity.
d. **Phospholipase** catalyzes the hydrolysis of lipids PhospholipaseA2, a well-studied component resent in the venom of all poisonous snakes.

It affects electron chain transport of mitochondria by inhibiting cytochrome C enzyme. RBC, WBC, platelets, vessel endothelium, muscle are damaged by this enzyme. Myoneural junction and peripheral nerves are also affected.

e. **Ophio amino acid** oxidase can activate proteases and peptides bound up in the cells. It hastens autolysis and putrefaction in viper venoms.
f. **Phosphodiesterase** found in snake venoms, has been found to produce immediate and profound fall in the systemic arterial pressure.
g. **Arginine ester hydrolase** present in crotalid and viper venoms, is coagulant in the effect and release bradykinin in the body.
h. **Acetyl choline** present in cobra and crotalid venoms is shown to have direct action on the heart and neuromuscular junction.

Hemotoxic effects of snake venoms[27]

Viper venom is purely vasculotoxic. It causes rapid swelling, necrosis, and eventually produce dry gangrene by thrombosis of the blood vessels supplies that area. Sytstemic absorption occurs through lymphatics and is slow produce lymphangitis. Coagulation disturbance typical to viper venoms, which cause complications and death.

Persistent bleeding from bite as well as from the cannula site indicates that coagulation mechanism has altered. Haemorrhage with altered capillary permeability produce pulmonary edema and profound shock. Renal injury shows signs and symptoms of loin pain, hematuria, oliguria which are common before death.

Haemorrhagins-1 & 2 are non-enzymatic component found in viper venoms. It rapidly causes lethal haemorrhage in many vital organs by destroying vascular endothelial lining and platelet functions.[15]

a. Coagulant property of venom is mainly due to activation of thromboplastin, factor X and factor V.
b. Anticoagulant effect is mainly from fibrinolysis, destruction of clotting factors.
c. Proposed mechanisms of viper venom induced coagulopathy include.[28] Venom toxin behaves thrombin-like activity and acting on fibrinogen leading to fibrin formation. Platelet aggregating effect of the venom.

Figure 11: a) Russell's Fang marks

Figure 11:b) Local bleeding from bite site

Figure : 12 (a) Haemorrhagic infarct of the anterior pituitary gland

Figure : 12 (b) Patient bitten a Burmese Russell's viper three years previously, showing signs of pan hypopituitarism: [loss of pubic and axillary hair and testicular atrophy.]

Neurotoxic effects of snake venom[29]

Krait, some type of cobras and coral snake cause mild local swelling. But naja naja produce painful local swelling, necrosis and blisters. Local necrosis produces putrid order due to direct cytolysis effect which look like “wet gangrene”. Skip lesion are unique. Systemic absorption occurs via venous route and cause neurotoxic symptoms.

The systemic signs and symptoms were nausea, vomiting, prostration, malaise, ptosis and opthalmoplegia herald the paralysis. Palsy of facial, neck, tongue and palatal muscles also noted. Death is due to respiratory failure.

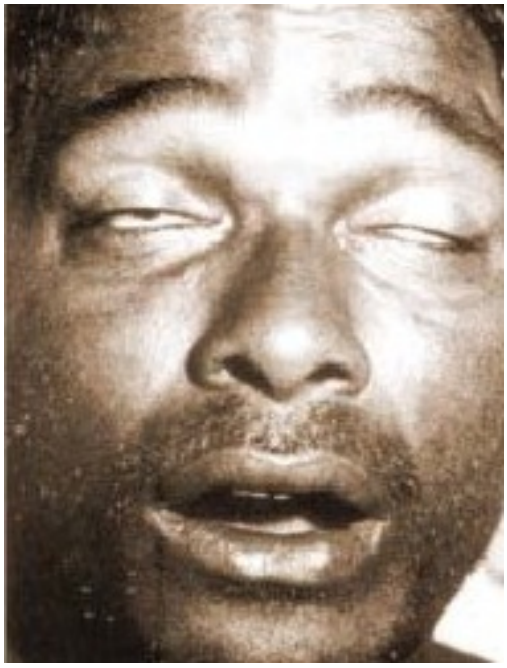


Figure: 12. Bilateral ptosis

Cardiotoxicity of snake venom[30, 31]

Cardiac complications are usually not a prominent feature. They are due to a direct acting cardio toxin where in the symptoms appear rapidly within 30 minutes to 2 hours. It has been reported to occur with both viper and elapid bites. Sudden hypotension, cardiac arrhythmias, cardiac arrest, peripheral circulatory failure and pulmonary oedema are the common clinical manifestations.[15]

Nephrotoxic effects of snake venom[32]

Acute renal failure (ARF) is characterized by fast and progressive decrease in renal function over a period of hours to days. Elevated plasma urea and creatinine concentrations confirm the diagnosis. ARF is usually associated with oliguria (24 h urine volume less than 400 ml) or anuria (24 h urine less than 100 ml), but this is not invariable. Some patients with ARF may have a normal urine output.

Acute renal failure is common in vasculo toxic snake bite with an incidence rate of 14% to 33% following R.Viper / Saw Scaled Viper. The mechanism for ARF is also not clear the possible reasons are haemolysis, haemorrhage, shock, myoglobinuria and coagulation defects.

Pathophysiology of Acute Renal Failure[33-35]

The exact pathophysiology of ARF following snakebite in not well known. This is due to the lack of a reproducible animal model.

Hypotension

Bleeding into tissue structures and external to the body with plasma loss produce circulatory collapse and hypotension. This is due to venom metaloproteinases which damage the basement membrane of vessel wall and disturb the integrity of endothelial cell lining.

In addition vasodilatation, unchecked capillary permeability, both are caused by direct/ indirect effect of venom. These two factors set a cascade of hemodynamic disturbance eventually leads to AKI.

Intravascular-haemolysis[36]

Intravascular haemolysis is due to activation of phospholipase A2. This direct lytic factor hydrolyse the RBC cell membrane proteins directly. This enzyme produce lysolecithin from serum lecithin, which also induce haemolysis.

Disseminated intravascular coagulation[37,38]

Homeostasis of coagulation system checked and balanced by cascade of vital reactions. This venom toxin interacts and disturbs these pathway and produce abnormal coagulation pattern.This is particularly seen in viper families[39].

The anticoagulant effect of venom is attributed to protein-c activation. This protein cleaves Va, VIIIa (clotting factors) and inhibit factors X and IX [40]. Directly-acting fibrinolytic proteins of the venom plays crucial role in the anticoagulation mechanism.

Zn-metaloproteinases (α/β fibrinogenases) is a main fibrinolytic enzyme involved in D.I.C. Platelet activated proteins is a toxin inhibit aggregation and stimulates activation of platelets.

D.I.C.is a salient feature of victims bitten by viperidae species[41]. The deposition of fibrin clot in microvasculature, glomerular capillaries, MAHA (micro angiopathic hemolytic anemia), thrombocytopenia with renal cortical necrosis shows the role of disseminated intravascular coagulation in pathogenesis of AKI[42, 43].

Direct Nephrotoxicity[44, 45]

Experimental studies in rabbits with venom toxins provide clues about renal glomerular lesions. The common histological changes are acute tubular/cortical necrosis. A good supportive evidence of direct nephrotoxicity is dose dependent decline of "inulin clearance" with "Fractional excretion of sodium" in an isolated perfused rat kidney. The prominent morphological structural lesions were found mainly in cortex. Glomerular epithelial, endothelial cell damage with ballooning and rupture of arterioles can be evident.[46,47]. Hypersensitivity reactions, sepsis and myoglobulinuria also induce renal injury. Myoglobulinuria commonly seen in sea snake bites resulting in muscle paralysis and necrosis.

Renal Histology

Acute tubular and cortical necrosis are the prominent histological features. Other types of glomerular injuries are insignificant.

Acute Tubular Necrosis (ATN) [48, 49]

ATN-an important lesion found in 60 to 80% of victims with AKI. Light microscopy shows dilated tubules lined with flattened epithelium. Severe damage to tubules is evident by epithelial cell necrosis, desquamation of the cells from the basement membrane. Brown pigment casts, hyaline casts seen in tubule lumen.

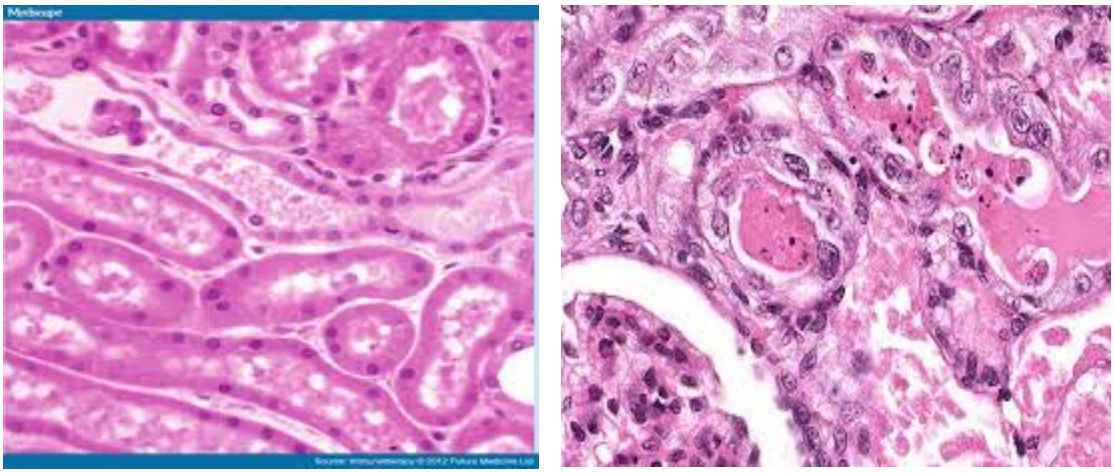


Figure 13a & 13b: Histology of Acute Tubular Necrosis

Inflammatory cell infiltration, oedema and haemorrhage are seen in interstitium. Intrarenal vessels are not involved. Distal, proximal tubular cells demonstrate dense intracytoplasmic inclusion bodies, dilated endoplasmic reticulum (smooth and rough). Apoptosis is severe in distal tubules while fibroblast hyperactivity is found in interstitium.

Granulated/degranulated form of eosinophils and mast cells are seen in vascular smooth muscle cells.

The salient features of acute tubular necrosis in snake bite are

a. Distal tubular apoptosis.
b. Fibroblast hyperactivity of interstitium.
c. Presence of eosinophils and mast cells in smooth muscle cells.
d. Predominant vascular injury.

Acute cortical necrosis[50, 51]

Bilateral patchy/diffuse cortical necrosis is seen in saw scaled viper bites. In India, cortical necrosis is quiet very common, but the reason is not clear. These patients have fibrin clots in the arterioles. A thin area below the subcapsular cortex escapes from necrosis, but area below this show severe necrosis. Hyperemia, leukocyte infiltration are seen in acute phase of necrosis, but calcification occurs in chronic stage. Organization of clot with fibroblastic proliferation occurs in healing process.

Glomerular Lesions[52,53]

Reports from few studies showed following types of glomerular changes can occur like proliferative glomerulonephritis, crescentic glomerulonephritis. These renal lesions have developed within 24-48 hours and ascribed to an allergic reaction to snake venom. Other glomerular changes observed are ballooning of arterioles, endothelial cells swelling, mesangiolysis and breach in glomerular basement membrane;. Immunofluorescence microscopy shows complement [C3], immunoglobulin [IGM] and fibrin clot deposits.[54]

Intense and Dense, mononuclear cell infiltratation in the interstitium, herald the acute interstitial nephritis.

Clinical Features

The clinical features of snake bite varied with

1. Gender and age of the person
2. Snake type
3. Location of the bite area
4. The amount and toxic nature of the venom

Persons with dry bite manifest unique clinical signs. Anxious person can hyper ventilate and develop dizziness. They also have sensory disturbance [pin & needles] and spasm in the extremities.

Some may develop vasovagal response with unconsciousness. Other develops anxiety and manifest wide range of wrong symptoms[46].

Mortality and Morbidity is high in extremes of age groups and also depend upon the physic of the casualty.

Local Effects[15]

Pain in bite location and few inflammations might be expected in all bites, the pain is most severe in viper bites. In case of elapid bites, patients often complain of burning sensation at the site. In the bitten limb, local increase in capillary permeability leads to swelling and bruising. Local tissue necrosis can occur either due to the toxic effect of the venom or to the complicating feature like arterial thrombosis, tourniquets. Regional lymphadenitis may also be noted.[55]

Secondary ulcers, chronic ulcers & osteomyelitis rarely occurs. Petechiae, ecchymosis, serous and hemorrhagic bullae are other local signs. Local manifestations usually present as cellulitis, edema, blister, erythema pain &tenderness with local rise of temperature at the bite area.

Systemic Effects

This can be divided into:

- Neurotoxic Effects
- Hemotoxic Effects
- Cardiotoxic Effects
- Nephrotoxic Effects
- Other complications such as shock

Neurotoxicity[34]

Confined to cobra, krait and sea snake bites, the symptoms of neurotoxicity appear from 20 minutes to 15 hours of the bite. According to the extent of envenomation, the symptoms may progress insidiously or rapidly to coma. Ptosis, varying degrees of ophthalmoplegia, palatal palsy, and pharyngeal paralysis with dysphagia, dysphonia, respiratory paralysis and flaccid limb paralysis are the common presentations with the advent of respiratory paralysis, hyoxaemia, drowsiness, convulsions and coma can ensue. The symptoms may progress slowly or fastlyto coma depending upon the envenomation spread.

Hemotoxicity

Hypofibrinogenemia and DIC are the main reason for in coagulation of blood and is the salient feature of systemic envenomation in wide spread geographical areas. Clinically, features of defective hemostasis include bleeding from the bite site and from cannula puncture sites, at other area of wound and from partially healed injuries in the body.[56,57] Common manifestations of spontaneous systemic bleeding includes, hematuria, gastrointestinal bleeding (hematemesis and melena), gingival bleeding, hemoptysis and cutaneous bleeding in the form of purpura and ecchymosis. Less common are menorrhagia and Central Nervous System bleeding. Subarachnoid hemorrhages / Intracranial hemorrhanges are most important reason for death. Pituitary and adrenal hemorrhage has also been reported. Bleeding may be prolonged and last as long as 26 days or more.[58] Other important feature is (DIC) which may either manifest as bleeding or thrombosis, with resultant ischemia. Intravascular hemolysis may also occur and result in hemoglobinuria and jaundice if it is massive. Hemotoxicity is said to be present if the patient's bleeding time is more than 8 minutes, prothrombin time more than 16seconds or the clotting time more than 30 minutes or if there is abnormal lysis of clot.

Nephrotoxicity[59, 60]

In victims with Acute Kidney Injury, oliguria / anuria frequently& quickly develops within the one day, but may be delayed till 48 to 72 hours after the snake bite. Few will have anuria, and rarely nonoliguric.

Urine analysis study might manifest microscopic or gross hematuria i.,e blood in Urine. Some victims experiences of pain in the loin with absence of urine output. This symptom indicates development of renal failure. Within next few days Blood pressure level considerably elevated with the absence of urine output and symptoms of kidney failure. Kidney failure symptoms (drowsiness, irritability, vomiting, hiccups, acidotic breathing, pericardial rub, convulsions) occur within 3 to 7 days of snake bites.[61]Nephrotoxicity diagnosed as acute renal failure when there is acute rise of serum creatinine to more than 1.5 mg /dl.

Other Manifestations

There may be features of increased capillary permeability which may appear within 24 hours of bite. These may be generalized, and lead to conjunctival, facial oedema, serous effusions in pleural cavity and abdominal cavity and radiological or clinical substantiation of pulmonary oedema. However, these are common following viper bites in Burma and Thailand and rarely seen in India. Rhabdomyolysis may rarely be seen and lead to generalized muscle aching, muscle tenderness and passage of dark red or black urine due to myoglobinuria. Arterial thrombosis, shock, necrosis of the limb, tetanus, and abortion in pregnant women are also seen after snake bite.[15]

Chronic complications[62]

Below complications may occur after snake bite

1. Tissue loss following infection and sloughing
2. Surgical wound debridement of the bite site
3. Amputation of affected part
4. Chronic non healing ulcer
5. Bone Infections [Osteomyelitis]
6. Neoplastic conversion might happened due to non-healing skin ulcers

Figure: 14a Physical disability&disfigurement due to poisonous snake bite

Figure: 14b Physical disability&disfigurement due to poisonous snake bite

Figure:15 Neoplastic conversion of a chronic skin ulcer with osteomyelitis few years after a bite by a poisonous viper.

Investigations

Laboratory investigations for snake bites envenomation have less sensitivity and specificity. They are actually useful to take decision for appropriate intervention[63].

There are two types of investigations as below

1. Specific
2. Non- Specific

1. Specific

(a) The 20-min whole blood clotting test (20 WBCT)

Simple and routinely use blood test for the diagnosis of coagulopathy is 20 minutes whole blood clotting time test. This is performed in a dry, clean glass tube which should not be washed with any cleansing agent.

Venous blood withdrawn from the patient is transferred to this tube and kept undisturbed for twenty minutes.

Blood should be clotted in twenty minutes if there is no envenomation. If not clotted it indicates coagulopathy and venom should from Viper[64]

(b) Enzyme linked immunosorbent assay (ELISA)

This test is very costly and available only in few sophisticated centres. It is mainly used to find out the species by using antigens of snake venom. So it is very useful in epidemiological surveys.

2. Non-Specific

a. Urine Analysis

It reveals

1. Albuminuria
2. Myoglobinuria
3. Hematuria
4. Hemoglobinuria

b. Complete Hemogram

1. HB – Pallor - > indicates Hemolysis
2. ↑PCV - > indicates hemoconcentration and plasma leak.
3. Leucocytosis [Neutrophils] - >Signifies systemic envenomation.
4. Thrombocytopenia

c. Prothrombin time

PT – Prolonged [Viper bite]

d. Activated partial thromboplastin time

aPTT – Prolonged [Viper bite]

e. Fibrin degradation products

FDP – Elevated

f. Renal Function Test

Bun and creatinine – Elevated - > indicate renal injury

g. ABG Analysis

h. Electrolytes

i. ECG

Useful in diagnosis of brady/tachyarrthymias

j. EEG

EEG changes are absorbed in nine out of ten victims shortly after the snake bite. These wave pattern arises from temporal lobes, and is not associated with development of encephalopathy.[68]

k. Blood Grouping & Typing

Snake Bite Management

World Health Organization along with S.E.A.R.O have released a protocol for the approach and management for snakebite induced envenomation[66]. These principles are accepted worldwide. The guidelines are published in the journal for Tropical Medicine and Public Health of South East Asia.[66]

First Aid[68]

The goal of first aid is to delay the systemic envenomation and abolish lethal sequealae by early transfer of victim to the medical care centre. All cultural taboos are discouraged because they worsen the situation. Somebody apply tourniquet as a first aid.

Give reassurance to the patient because most of the bites are dry. Apply splints and immobilize the affected part and finally transport the victim to medical centre.

PIM

Pressure immobilization method **(PIM)** has primarily developed and adapted by the University of Melbourne in Australia (venom research unit). This technique encourages immobilization of the affected part by applying splint or bandages. Once the splint has applied, the part should not be mobilized. Otherwise, the technique is ineffective. Health education to health workers, community health providers, and civilians is essential regarding early immobilization and safe transfer of patient to the hospital.

Hospital Management

In the emergency department, the patient should be assessed for airway, circulation, breathing, and level of consciousness. Immediate attention to be given to patient with shock, respiratory failure, and with altered sensorium/unconsciousness. O2 is mandatory to all patients with envenomation. IV access with wide bored cannula is also important. Isotonic fluid like RL/NS should be infused to prevent shock.

History

First confirm whether the victim had a snake bite or some other bite made by other animals. Once you confirmed, then search for the fang marks and local signs of envenomation. History about time interval between the bite and hospital admission is essential. Other detailed history regarding comorbidity, allergy, adverse drug reactions, systemic illness also essential.[6] If the patient had brought the snake, careful examination and identification is important. A very quiet interesting is crotalids envenomate even after death.

General Examination[6]

Patient consciousness, orientation and vitals should be assessed first. Then, examine the bite area and search for local signs like pain, swelling, bleeding, and cellulitis. Limb circumference should be calculated at frequent intervals to assess the progression of cellulitis.

The list given below is useful to assess the severity of envenomation

1. Snake type – venomous/nonvenomous.
2. Rapid progression of cellulitis.
3. Palpable, tender, lymph node enlargement, draining the affected part.
4. Rapid onset of systemic symptoms.
5. Spontaneous and massive bleeding.
6. Altered urine colour (hemoglobulinuria).
7. Absence of peripheral pulses (compartment syndrome).

Specific Therapy

Anti snake Venom[68]

Anti snake venom is the specific antidote for venom of snakes. It is an immunoglobulin derived from immunized equine serum. First venom is given to the horse and immunized, then immunoglobulin's are extracted and purified.

Figure 16 ASV Vial

Two types of ASV are available.

1. Monovalent.
2. Polyvalent.

The main disadvantages of monovalent ASV are price, less availability, and it require species confirmation[73]. The advantages of polyvalent ASV are cheaper, wider availability, and Para specific activity (antibodies developed against venom of one type of snake neutralize the other type of snake venom).

There are two forms of ASV available. 1. Liquid form 2. Lyophilized form

1. Liquid form – Cold chain is mandatory, cost effective, high chances of physicochemical changes.
2. Lyophilized form – Cold chain is not required, costly, and stable. Lyophilized form is reconstituted by the method of swirling with 10 ml of N.S.

Manufacturing Units in India

1. Central Research Institute – Himachal Pradesh (Kasauli).
2. Haffkine Corporation – Maharashtra (Mumbai).

THE CROFAB (crotalidae immune Fab)

FDA has recently approved the use of CROFAB for the crotalidae envenomation. This crofab binds with fab site of IgG immunoglobulin. This immune fab acts in three ways. One is to neutralize the toxin. The second one favour the redistribution of venom from the affected tissues and the final one facilitate its elimination from the body circulation. The incidence of acute and late allergic responses to this product is comparatively lower than the regular antivenom.

Indications For ASV[73]

Antivenom therapy is indicated with every patient who shows severe systemic and local toxicities. ASV administration is associated with high chances of adverse reactions. It is costly and is not very well available in many areas, so pros and cons to be weighed before initiation of treatment.

1. **Systemic Toxicity**[74, 75]
    a. Haematological abnormalities – WBCT more than 20 minutes, spontaneous bleeding, low platelet count.
    b. Neurotoxicity -Ophthalmoplegia, bulbar palsy, etc.
    c. Cardiovascular- Hypotension, acute pulmonary oedema, circulatory collapse, dysrhythmia.
    d. Severe rhabdomyolysis.
    e. Acute kidney injury.
2. **Local Envenomation**
    a. Rapidly evolving cellulitis.
    b. Cellulitis affecting more than half of the involved extremities.
    c. Severe bruising and blistering.
    d. Victims bitten by spices which cause severe local necrosis.

Contraindications for ASV[66]

There are no relative or absolute contra indications for ASV administrations, though, it is used very carefully by a person who has atopic history and showed immune response to equine antisera. Pregnancy is not a contra indication.

Antivenom [ASV] Therapy

It is ideal to start ASV before four hours from the time of bite, ASV can effective within a day of bite.

Dose of ASV [76]

Till now there is no evidence based on clinical trials for ASV dosage. In India we determine the dosage depends on the severity of envenomation.

Figure 17 Milking of Venom

Regional Office of South East Asia for World Health Organization strongly suggest the quantity of antivenom to offset based on the average venom yield by a captive snakes are milked.[71] In India we determine the dosage depends on the degree of envenomation. Usually Russell's viper delivers approximately Sixty Three milligrams of venom with the standard deviation of plus or minus Seven milligram per maiden bite. [77,78]

One polyvalent vial neutralizes Six milligram of Russell's Viper Venom, so each and everyone should be given Eight to Ten vials primarily. There is no dosage difference between Child and Adult. Because snakes always delivers same quantity of venom.[66]

Response is evident by reversal of normal BP.[80] Bleeding arrest should happen within half an hour, reversal of coagulopathy occurs more or less six hours to regularize. However Neurotoxicity symptoms improves within half an hour but it may take 1 to 2 days for complete retrieval.[81]

Repeated dosage of ASV must be given if there is irreversibility of coagulopathy persistence more than six hours or bleed continuously beyond one to two hour of the initial dosage. It is necessary to repeat the dosage if signs of neurotoxic or cardiotoxic are worsening even after the one to two hours.[66]

ASV Administration

ASV administration can handle in 2 different ways.

1. Intravenous infusion – Five to Ten milligram per Kg body weight of diluted antivenom
2. Slow Intravenous infusion – Dosage at a rate of 2 ml per minute. All patients has to be monitored for a period of minimum 60 minutes for progress of anaphylaxis

ASV should not be injected directly in the snake bitten place, because this kind of administration develop major risk like intolerable pain and increase pressure in intra compartmental region.

Intramuscular injections are not advisable, because it has large molecules. This large molecule spreads slowly thorough lymphatics and impaires the bio availability.

This route of administration also causes sciatic nerve injury and hematoma formation. [66] Injection adrenaline should be kept ready before ASV administration.

ASV Sensitivity Testing

ASV sensitivity testing is not at all recommended, because not able to foresee the development of either early reactions which is anaphylactic or late reactions which is Serum sickness.[66]

This testing allows the patient for the exposure of serum protein with development of reaction further delays the treatment.

ASV Reaction

There are two types of ASV reaction, they are as below

a. **Early**
b. **Late**

ASV does not develop any reaction for approximately 75-80% of patients, i.e it develop reactions to the 20-25% of patients. Approximately 20% patients treated with ASV develop either early or late reaction.[66]

Early Reactions

Early reactions happens within One Hundred and Eighty minutes from the time of treatment started and it develops below characterization

1. Itching.
2. Urticaria.
3. Dry Cough.
4. Nausea & Vomiting.

5. Abdominal Colic.
6. Diarrhea.
7. Tachycardia.
8. Fever.
9. Hypotension.
10. Bronchospasm.
11. Angioedema.

Late Reactions

Late reactions develop between one and twelve days [Average 7 Days] from the date of treatment. Clinical features are

1. Fever.
2. Nausea.
3. Vomiting.
4. Diarrhea.
5. Itching.
6. Recurrent Urticaria.
7. Arthralgia.
8. Myalgia.
9. Lymphadenopathy.
10. Immune complex nephritis.
11. Encephalopathy [Very Rarely].

Pyrogenic Reactions

It happens due to manufacturing defects or contamination of Antivenom by pyrogens, normally it become evident within two hours from the time of treatment. Symptoms are as below

1. Chills and rigors.
2. High grade fever.
3. Hypotension.

Treatment For ASV Reaction

Treatment for ASV reaction is below **[Early]**

1. Adrenaline
2. Antihistamine
3. Glucocorticoid

Adrenaline

- Adult Dosage - 0.50 milligram [One in Thousand dilution] - > Route – intramuscular
- Child Dosage - 0.01 milligram per Kilogram body weight - > Route - intramuscular

Antihistamine [Chlorpheniramine maleate]

- Adult Dosage - 10 milligram - > Route - intravenous
- Child Dosage - 0.02 milligram per Kilogram body weight - > Route – intravenous

Glucocorticoid [Hydrocortisone]

- Adult Dosage - 100 milligram - > Route - intravenous
- Child Dosage – 2 milligram per Kilogram body weight - > Route – intravenous

Treatment for ASV reaction is below **[Late]**

1. Antihistamine
2. Glucocorticoid

Antihistamine [Chlorpheniramine maleate]

- Adult Dosage - 2 milligram every six hours - > Route - Oral
- Child Dosage - 0.25 milligram per Kilogram body weight divided dose - > Route – Oral

Glucocorticoid [Prednisolone]

- Adult Dosage - 5 milligram every six hours - > Route - Oral
- Child Dosage – 0.7 milligram per Kilogram body weight divided dose- > Route – Oral

Supportive Treatment

The Patient, who envenomed severely, should be admitted in Intensive Care Unit. Patients who have not severely envenomed can be admitted in the ward and monitoring to be happened closely.

Patients who envenomed severely can be identified from the below any of the symptoms

a. Coma.
b. Respiratory paralysis.
c. Hypotension.
d. Pulmonary edema.
e. Syncope.

Supportive therapy is essential buy time till the time recovery of the damaged organs.

Different types of supportive methods as summarized below

Management for Coagulopathy[71]

ASV plays crucial role in the correction of Coagulopathy. Once ASV has given Coagulopathy immediately reverses. In unique cases with severe uncontrolled bleeding, the following can be given to reverse the Coagulopathy.

1. Fresh whole blood
2. Cryoprecipitate
3. Fresh frozen plasma
4. Platelet concentrates

Management for Renal Failure [66]

Renal failure is more common in victims who are envenomed with viperiade family. Renal perfusion can be improved by following ways.

a. IV fluids – Isotonic saline
b. Diuretics – Furosemide (up to 100 milligram)

c. Dopamine – 2 microgram per Kg per minute

Once patient develops oliguria for the period of more than 6 hours or anuria the following should be done.

1. Central venous catheter insertion.
2. Urethral catheterization.
3. Fluid balance monitoring.

If myoglobinuria / hemoglobinurea developed the following should be done to prevent Kidney damage.

1. Isotonic IV fluids [Hypovolemia]
2. Injection mannitol [Urine Production]
3. Bicarbonate [Acidosis]

Management for Neurotoxicity[82]

Even though antivenom therapy is indicated, patient with bulbar palsy as well as respiratory failure should be given other line of treatment. The earlier signs of severe neurotoxicity are as follows

a. Slurring of speech.
b. Regurgitation of fluids.
c. Poor Cough response.
d. Motor Paralysis.
e. Poor respiratory effort.

The patient with the above symptoms should be intubated and supported with mechanical ventilation. The Neurotoxins affects the Myoneural junction and its activity. Its activity is reversed by anticholinesterase.

Anticholinesterase [Neostigmine] [82]

- Initial Dose - 0.5 milligram – Every 30 minutes – Route - > Intra venous – 5 Doses
- Repeat dose - 0.5 milligram – gap of 2 -12 Hours - Route - > Intra venous

Atropine[82]

Dose – 0.6 milligram – Route - > IV [before each time of Neostigmine]

Disanayake et al[82] did a prospective study in Neurotoxin envenomation and they recommend Neostigmine in two situations.

a. Positive Tensilon test
b. Decremental response to repetitive nerve stimulation.

Management of cellulitis[83]

1. Limb elevation and immobilization
2. Cellulitis has been treated with broad spectrum of antimicrobial agents which covers gram positive and gram negative organisms and anaerobic agents.
3. Fasciotomy should be considered with the development of compartment syndrome. The signs and symptoms of compartment syndrome are vague. So diagnosis is difficult. However we can diagnosis by monomeric technic. 22 gauge needle is inserted at the affected site and it is connected to the manometer device. If pressure is more than 55 centimeter of H_2O diagnosis is confirmed.
4. Fasciotomy might be delayed until reversal of coagulopathy symptoms, otherwise it endangers the life. Tetanus Toxoid.[71]

MATERIALS AND METHODS

Study Group:

100 Snake bite patients admitted in Medical ward, Government Vellore Medical College Hospital, Vellore.

Study Design:

Clinical, prospective and observational study

Study Population:

This study was conducted among 100 patients who were admitted in medical ward Govt. Vellore medical college hospital, Vellore.

Duration of Study:

1 year (August '2014– July'2015)

Inclusion Criteria:

1. Features suggestive of snake bite with or without local and systemic envenomation.
2. Definite fang marks noted.
3. Patients or attenders have seen the offending snake.
4. Progressive increase in serum creatinine more than "0.3mg/dl" from baseline, a percentage increase in the serum creatinine concentration of more than 50% (or) oliguria of less than 0.5ml per kg per hour for more than 6 hours.

Exclusion Criteria:

Patients with history of suspected snake bite, where in

1. Patient or attenders have not seen the snake.
2. Patient with pre-existing renal disease.
3. Patient with risk factors for developing renal diseases with history of snake bite (Diabetes mellitus, systemic hypertension, connective tissue diseases)

Data Collection:

All patients confirmed of snake bite will be selected and subjected to detailed history, physical examination and biochemical test with special reference to renal involvement. Special tests like bleeding time, clotting time, serum electrolytes and USG abdomen was done whenever necessary. Systolic Blood pressure <90mmHg is considered as hypotension.

Patients were followed up till discharge or death. They were treated accordingly. Clinical data was tabulated. Statistical analysis was done.

Statistical Analysis:

All statistical analysis were performed using the SPSS (Software package used for statistical analysis) package. A “P” value of less than 0.05 was considered to be statistically significant.

Collaborative Department:

BIOCHEMISTRY & NEPHROLOGY

Consent:

Individual written and informed consent

Ethical Clearance:

Obtained from “ETHICAL COMMITEE” Government Vellore Medical College, Vellore

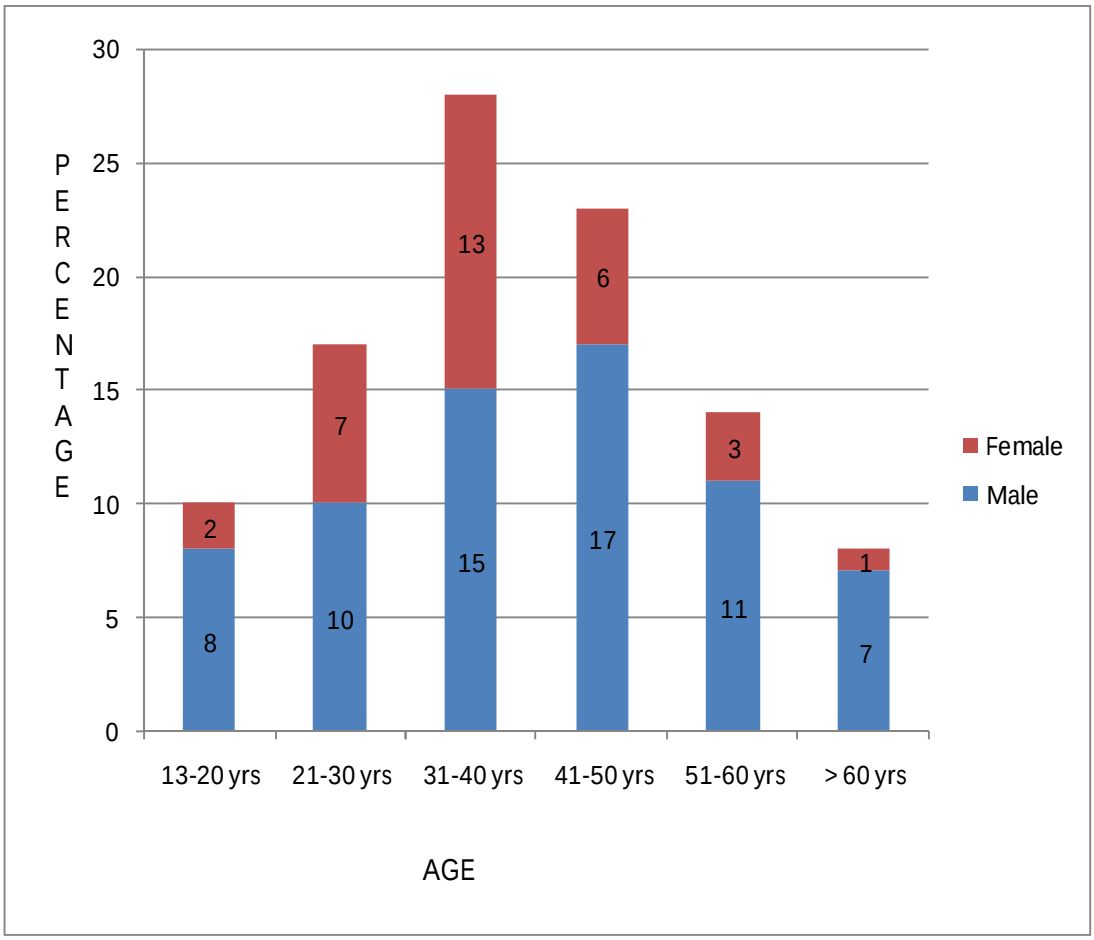
RESULTS

A prospective clinical study of 100 patients of snake bite from August 2014 to July 2015 was done to know the clinical profile and outcome in GVMCH, Vellore.

TABLE 1 : AGE AND GENDER DISTRIBUTION

Age	Male		Female	
	Number	Percentage	Number	Percentage
13-20 yrs	8	11.7%	2	6.3%
21-30 yrs	10	14.7%	7	21.8%
31-40 yrs	15	22.1%	13	40.6%
41-50 yrs	17	25%	6	18.8%
51-60 yrs	11	16.2%	3	9.4%
> 60 yrs	7	10.3%	1	3.1%
TOTAL	68	100%	32	100%

FIGURE 18: AGE AND GENDER DISTRIBUTION



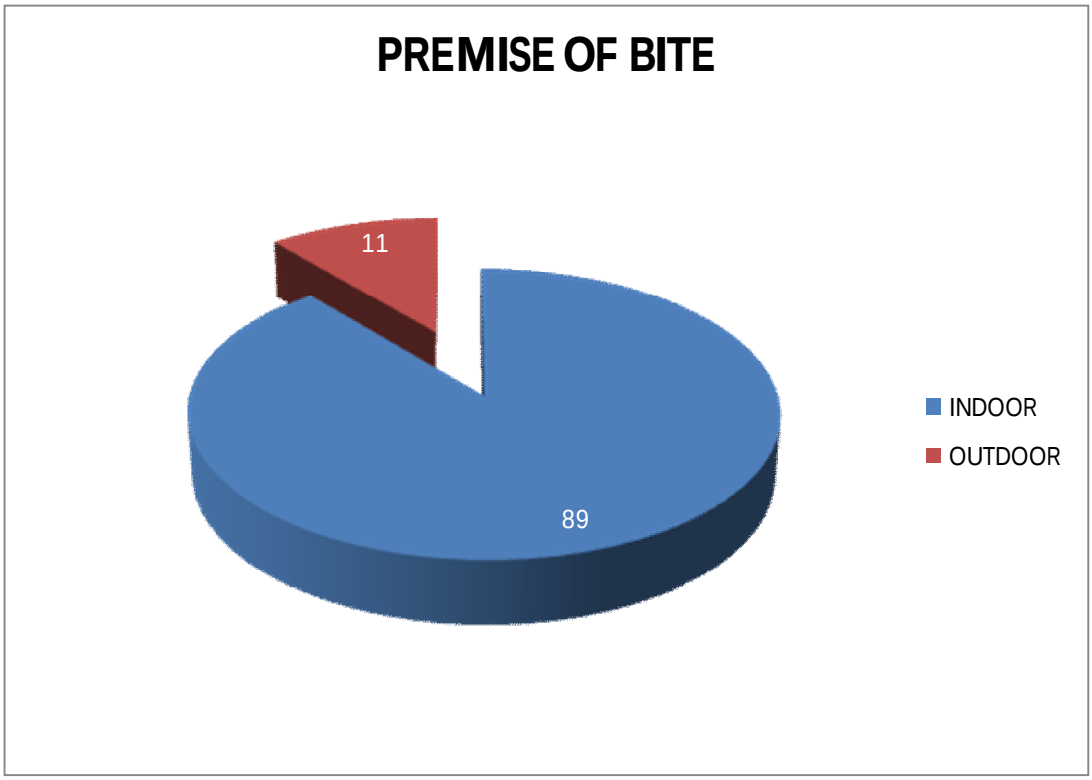
Maximum incidence of snake bite occurred in the age group between 31 and 40 years (28%) followed by 41-50 years (23%).

Males are more commonly affected (68%) than females (32%).

TABLE 2: PREMISE OF BITE

PREMISE OF BITE	NUMBER
INDOOR	89
OUTDOOR	11

FIGURE 19: PIE CHART SHOWING PREMISE OF BITE

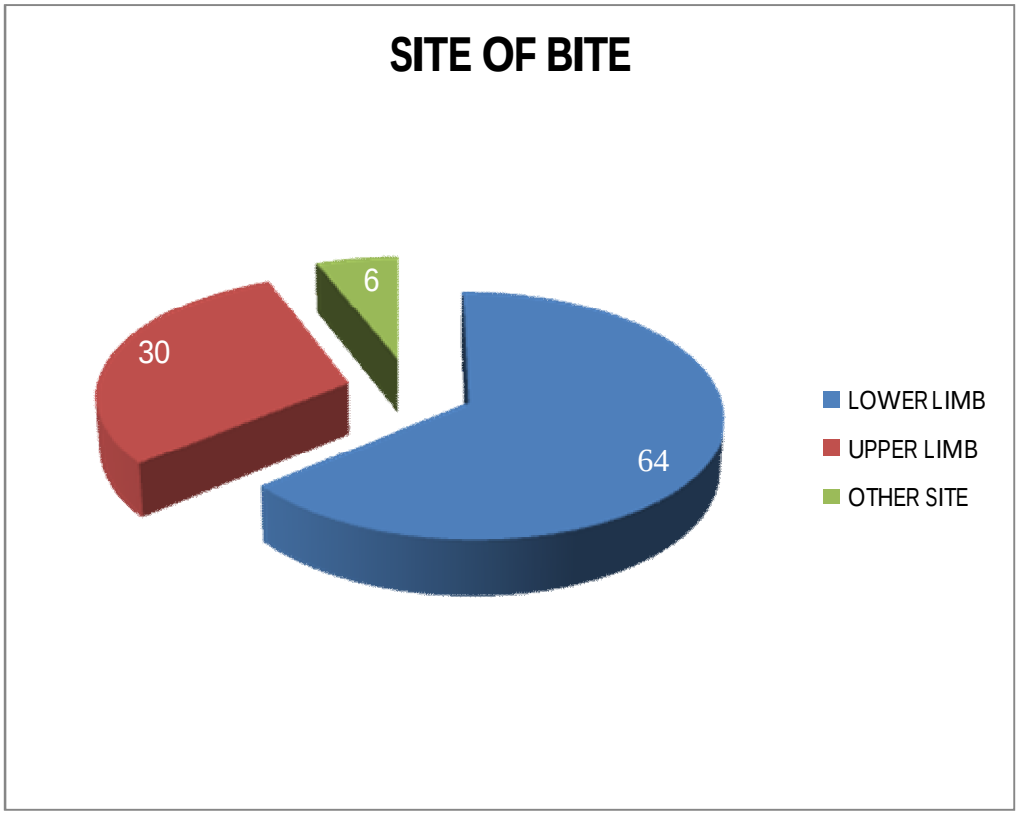


Most of the bites occurred in outdoor. (89%)

TABLE 3: SITE OF BITE

SITE OF BITE	NUMBER
LOWER LIMB	64
UPPER LIMB	30
OTHER SITE	6

FIGURE 20: PIE CHART SHOWING SITE OF BITE

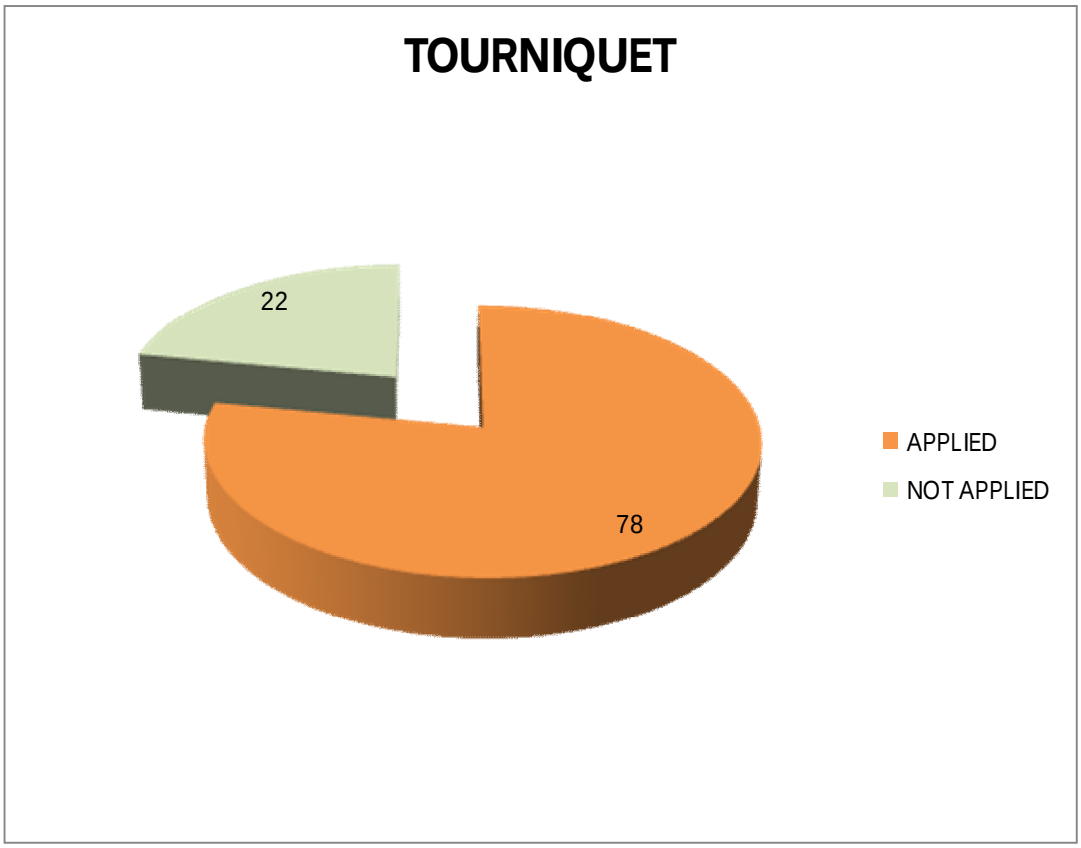


Lower limb (64%) is the most common site for snake bite followed by Upper limb (30%) and other sites (6%).

TABLE 4: APPLICATION OF TOURNIQUET

TOURNIQUET	NUMBER
APPLIED	78
NOT APPLIED	22

FIGURE 21: PIE CHART SHOWING APPLICATION OF TOURNIQUET

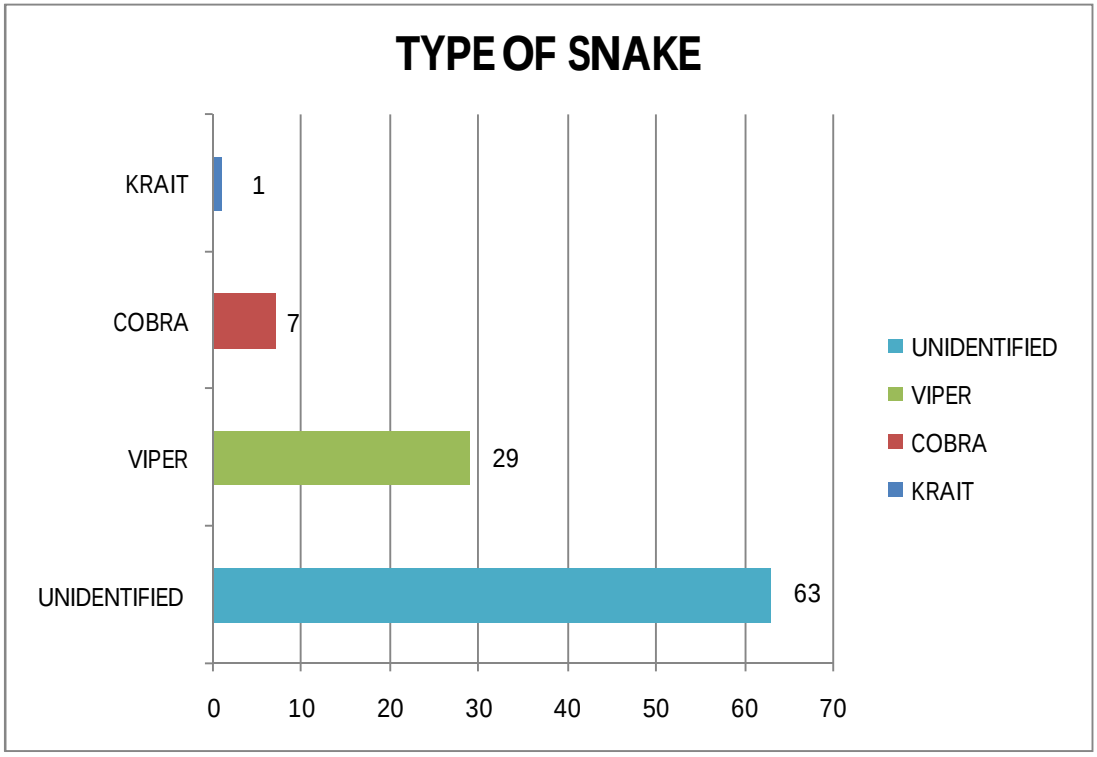


Majority of the victims had applied tourniquet. (78%)

TABLE 5: TYPE OF SNAKE

TYPE OF SNAKE	NUMBER
UNIDENTIFIED	63
VIPER	29
COBRA	7
KRAIT	1

FIGURE 22: BAR DIAGRAM SHOWING TYPE OF SNAKE

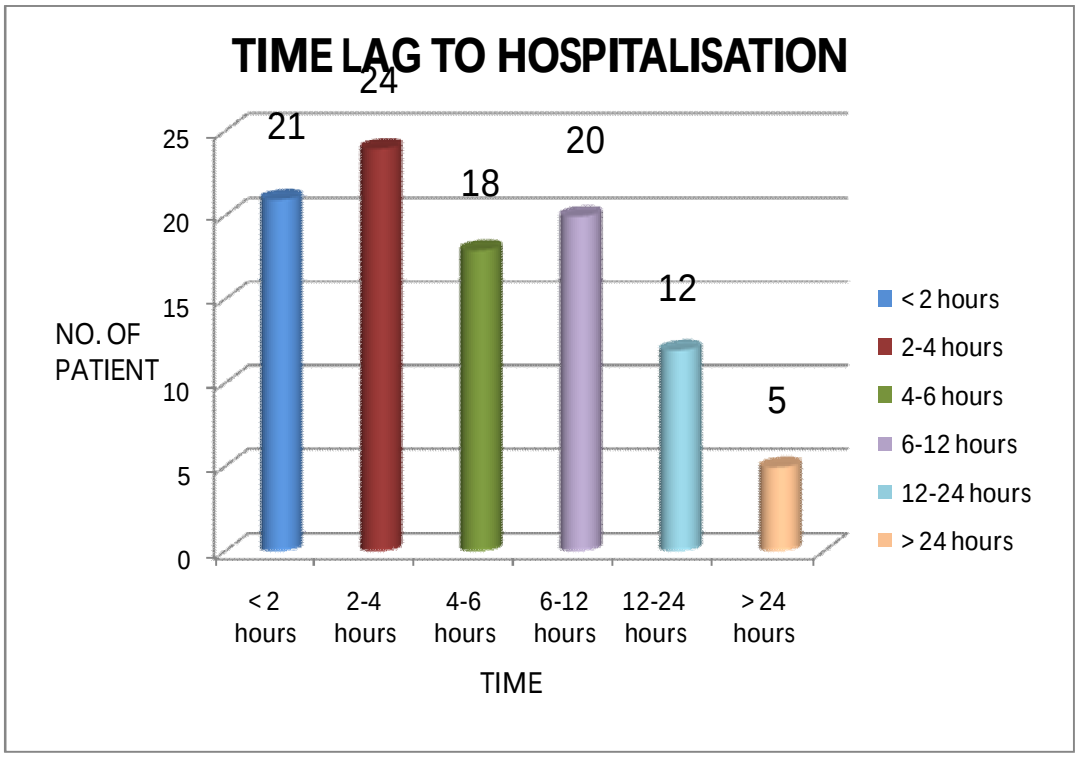


Among 37 cases in which snake was identified, bites due to Viper (29) were most common followed by Cobra(7) and Krait(1). 63 patients could not identify the snake.

TABLE 6: TIME LAG IN HOSPITALIZATION OF PATIENTS

TIME LAG TO HOSPITALISATION	NUMBER
< 2 hours	21
2-4 hours	24
4-6 hours	18
6-12 hours	20
12-24 hours	12
> 24 hours	5

FIGURE 23: BAR DIAGRAM SHOWING TIME LAG IN HOSPITALIZATION OF PATIENT

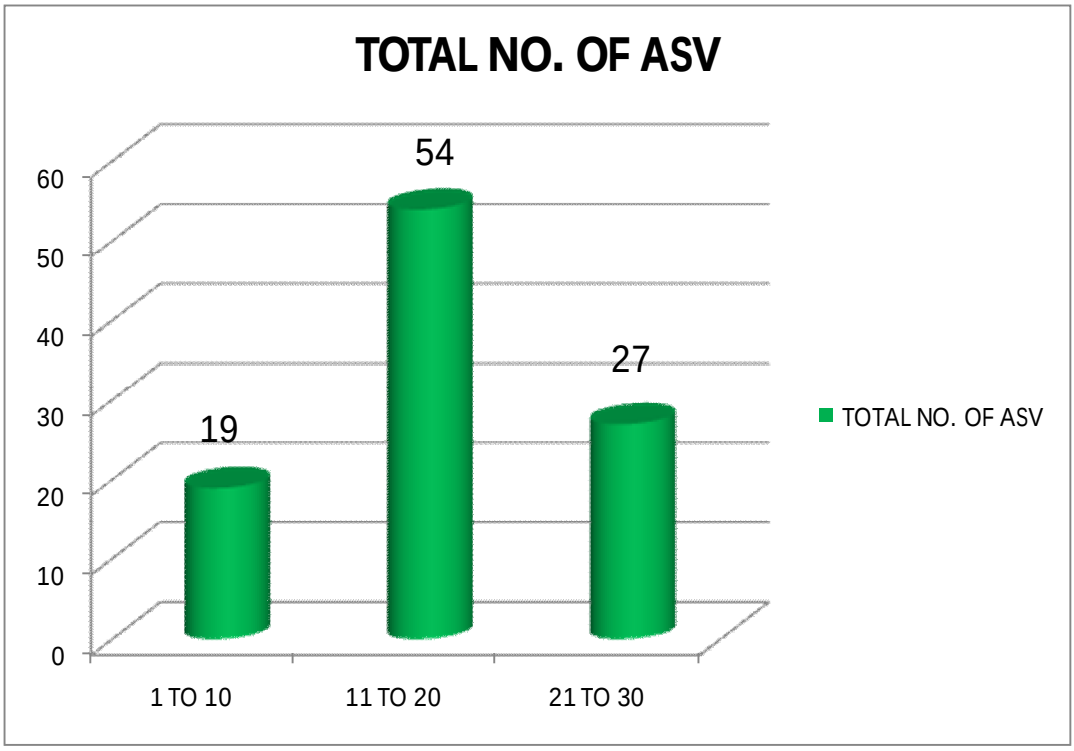


Majority of patients reported within 4 hours of the bite (45). 18 patients presented within 4-6 hours while 32 patients presented within 6-24 hours. 5 patients presented after 24 hours of the bite.

TABLE 7: TOTAL NUMBER OF ASV IN VIALS

ASV IN VIALS	NUMBER OF PATIENTS
1 To 10	19
11 To 20	54
21 To 30	27

FIGURE 24: TOTAL NUMBER OF ASV IN VIALS

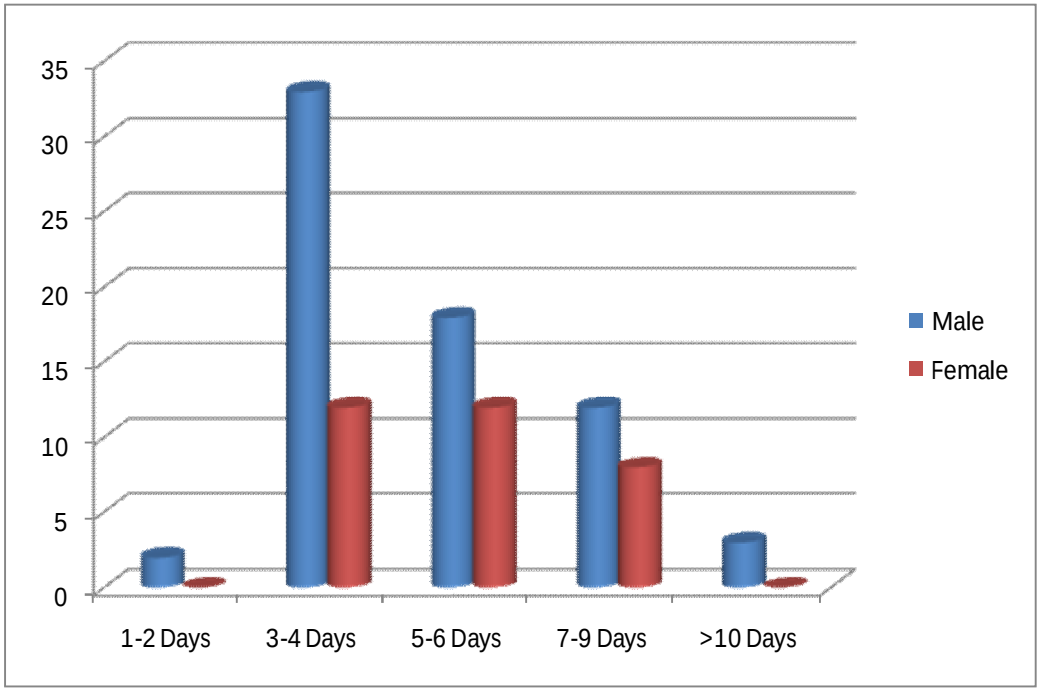


54 patients received about 11-20 vials and 27 patients received 21-30 vials rest of 19 patient received 1-10 vials

TABLE 8: DURATION OF STAY IN WARD

Duration of stay in days	Male	Female	Total
	No	No	
1-2	2	0	2
3-4	33	12	45
5-6	18	12	30
7-9	12	8	20
10 & above	3	0	3
Total	68	32	100

FIGURE 25: DURATION OF STAY IN WARD

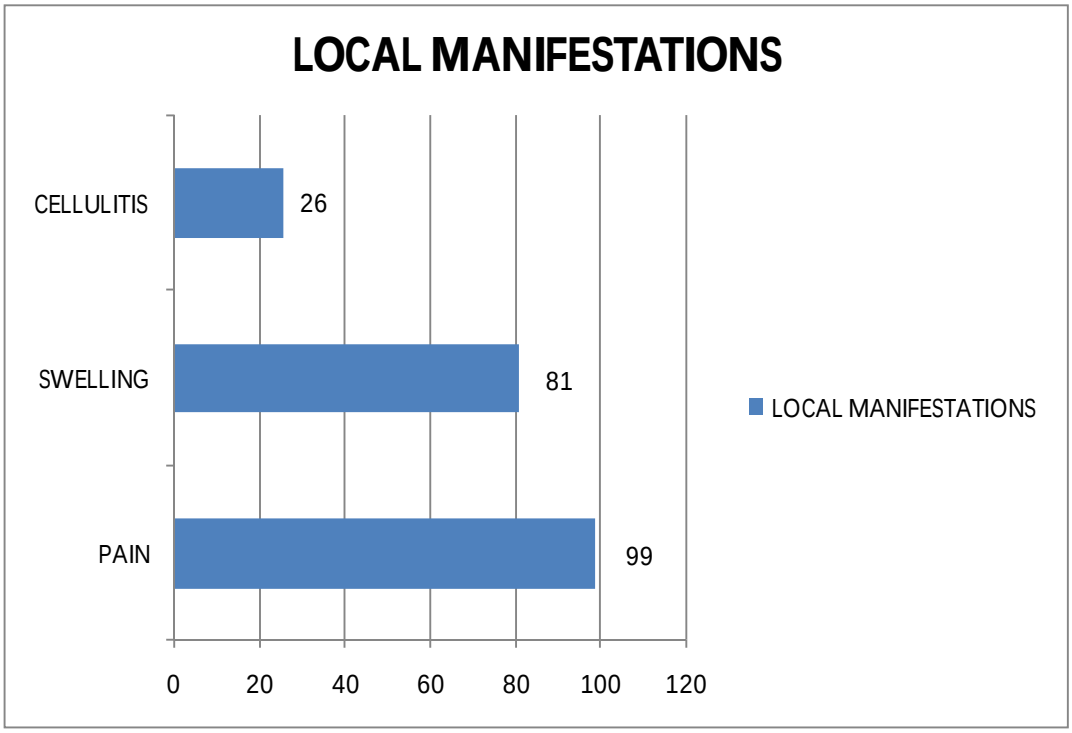


Majority of patients stayed in the hospital for 3 to 4 days (45%) and only 3% stayed in the ward more than 10 days.

TABLE 9: LOCAL MANIFESTATION OF ENVENOMATION

Local Manifestation	Male	Female	Total
	No	No	No
Pain	68	31	99
Swelling	55	26	81
Cellulitis	17	9	26

FIGURE 26: LOCAL MANIFESTATION OF ENVENOMATION

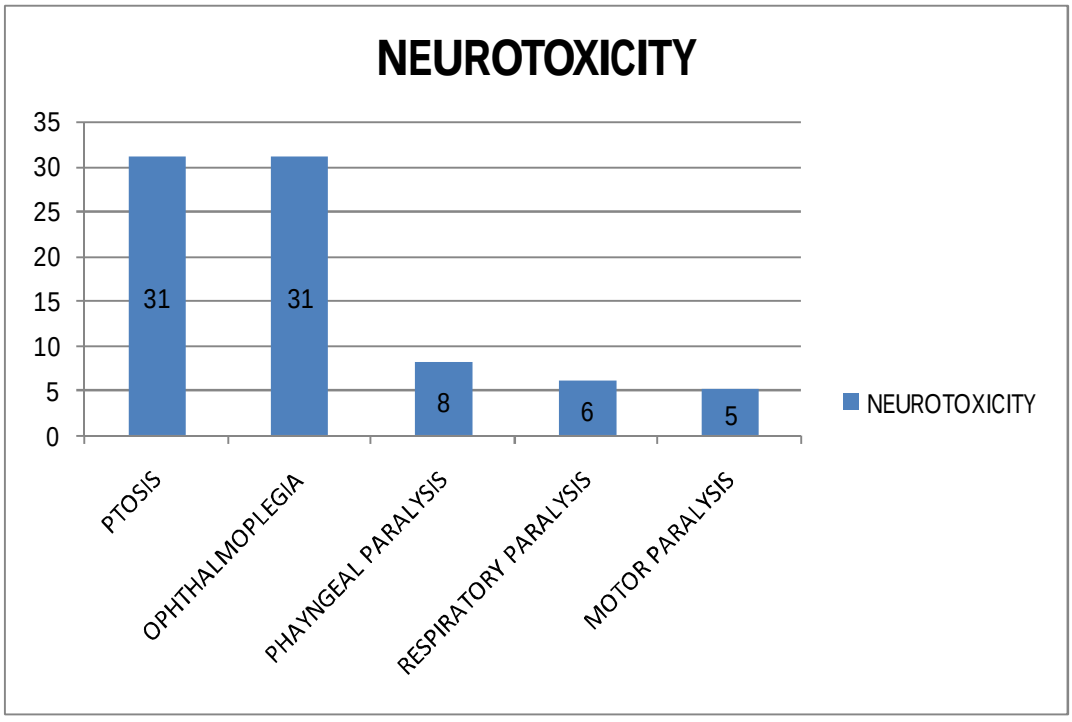


Among local manifestation (99%) of patient experienced pain followed by swelling (81%) and cellulitis (26%)

TABLE 10: NEUROTOXIC MANIFESTATION OF ENVENOMATION

Neurotoxic Manifestation	Male	Female	Total
	No	No	No
Ptosis	22	9	31
Ophthalmoplegia	22	9	31
Pharyngeal paralysis	7	1	8
Respiratory Paralysis	4	2	6
Motor Paralysis	5	0	5

FIGURE 27: NEUROTOXIC MANIFESTATION OF ENVENOMATION

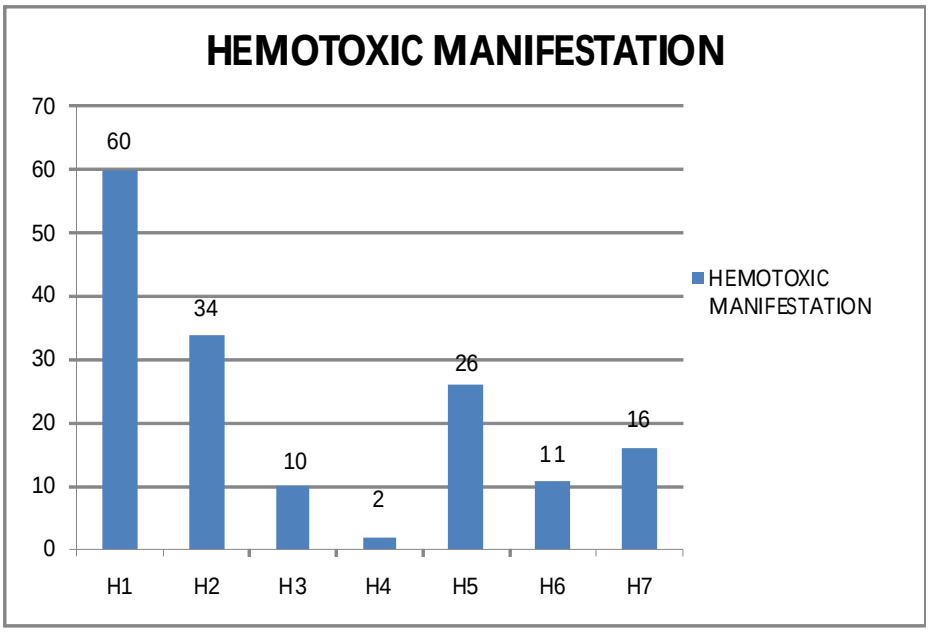


Among 100 patients of snake bite, 81 patients developed neurotoxicity, 31 patients developed ptosis, 31 patients developed of opthalmoplegia and 6 patients developed respiratory paralysis.

TABLE 11: HEMOTOXIC MANIFESTATION OF ENVENOMATION

Hemotoxic Manifestation	Male	Female	Total
	No	No	No
Bleeding from bite site (H1)	41	19	60
Mucosal Bleeding (H2)	23	11	34
Epistaxis (H3)	8	2	10
Petechiae (H4)	2	0	2
Hematuria (H5)	19	7	26
Hematemesis (H6)	8	3	11
Hemoptysis (H7)	10	6	16

FIGURE 28: HEMOTOXIC MANIFESTATION OF ENVENOMATION

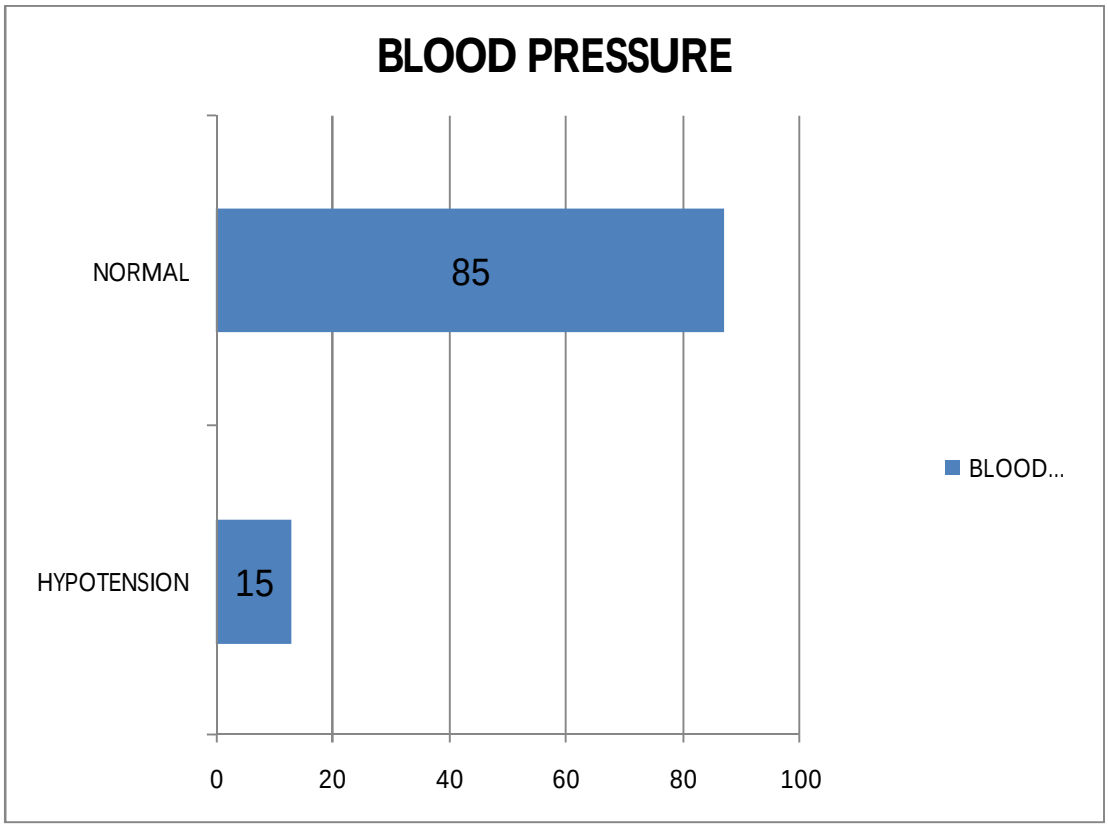


60 patients developed bleeding from bite site, followed by 34 patients developed mucosal bleeding. 26 patients presented with hematuria.

TABLE 12: BLOOD PRESSURE VALUE

BLOOD PRESSURE	Total	%
Normal	85	85
Hypotension	15	15

FIGURE 29: BLOOD PRESSURE VALUE

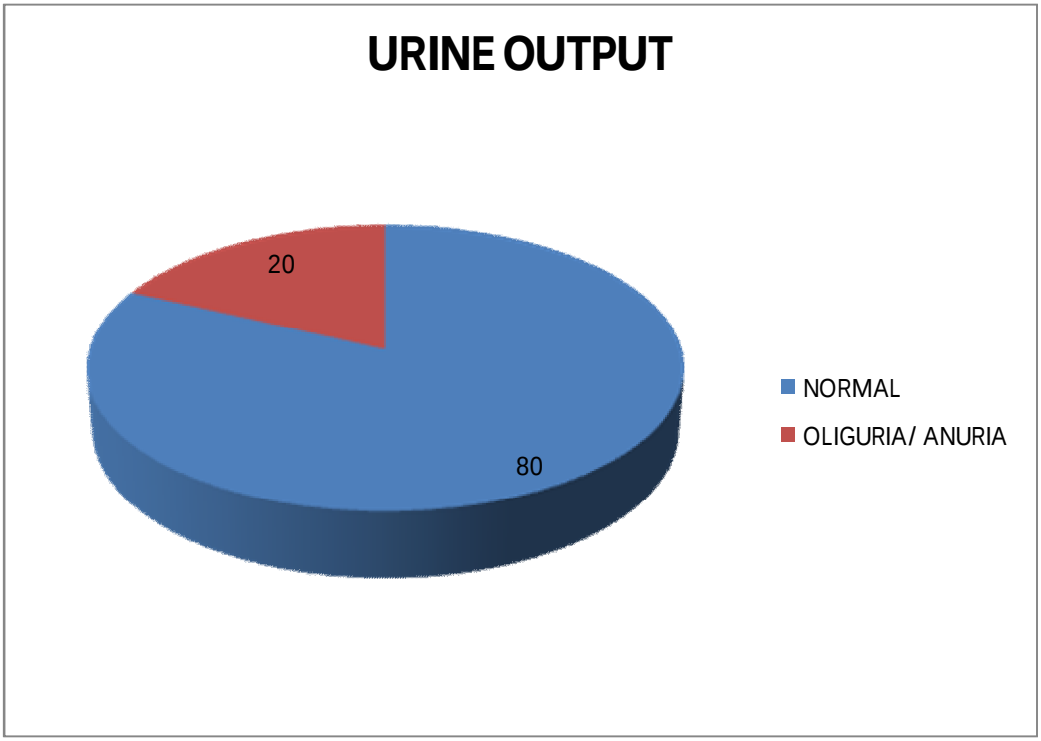


Among 100 cases 15 cases developed hypotension

TABLE 13: URINE OUTPUT

URINE OUTPUT	Male	Female	Total
	No	No	
Normal	53	27	80
Oliguria/Anuria	15	5	20

FIGURE 30: URINE OUTPUT

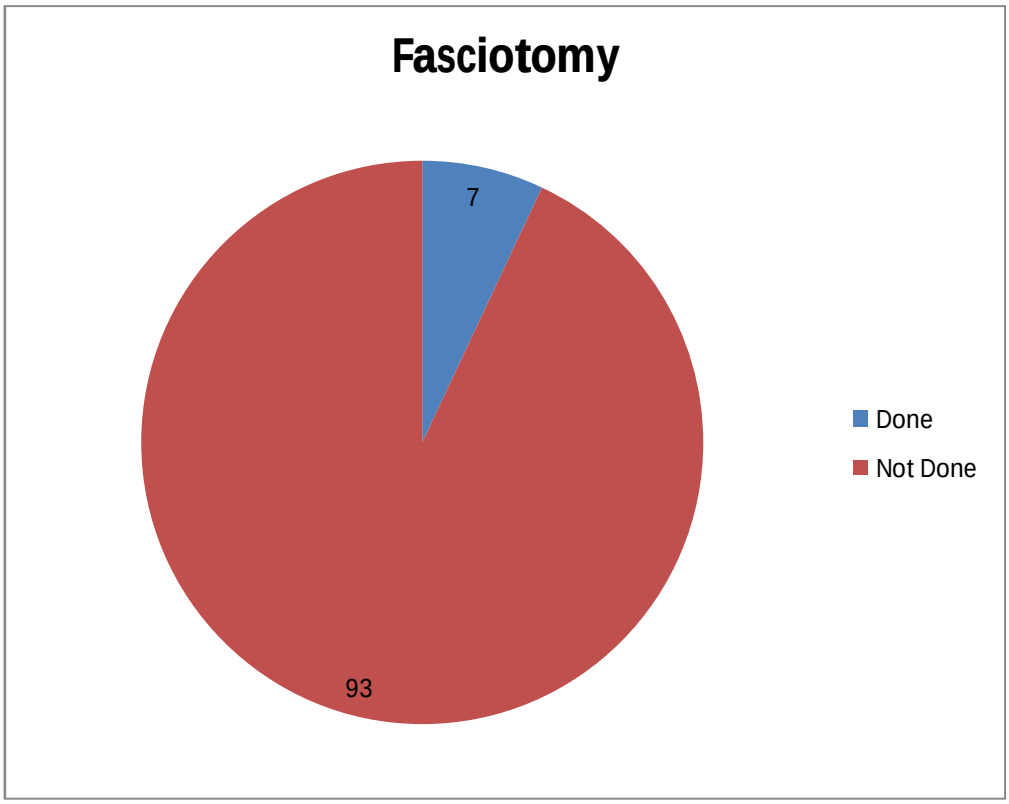


Among 100 patients 20 patients developed oliguria.

TABLE 14: PATIENT NEEDED FASCIOTOMY

Fasciotomy	Number	Percentage
Done	7	7
Not Required	93	93

FIGURE 31: PATIENT NEEDED FASCIOTOMY

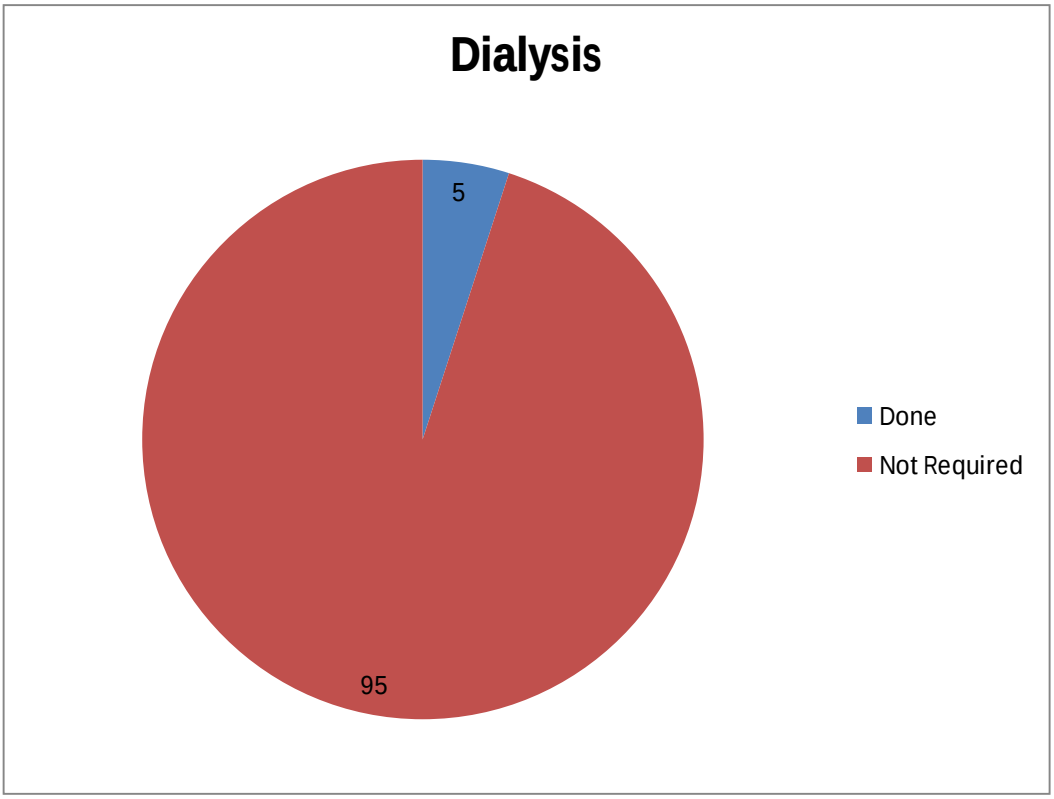


Among 100 patients 7 needed fasciotomy.

TABLE 15: PATIENTS NEEDED DIALYSIS

Hemodialysis	Number	Percentage
Done	5	5
Not Required	95	95

FIGURE 32: PATIENTS NEEDED DIALYSIS

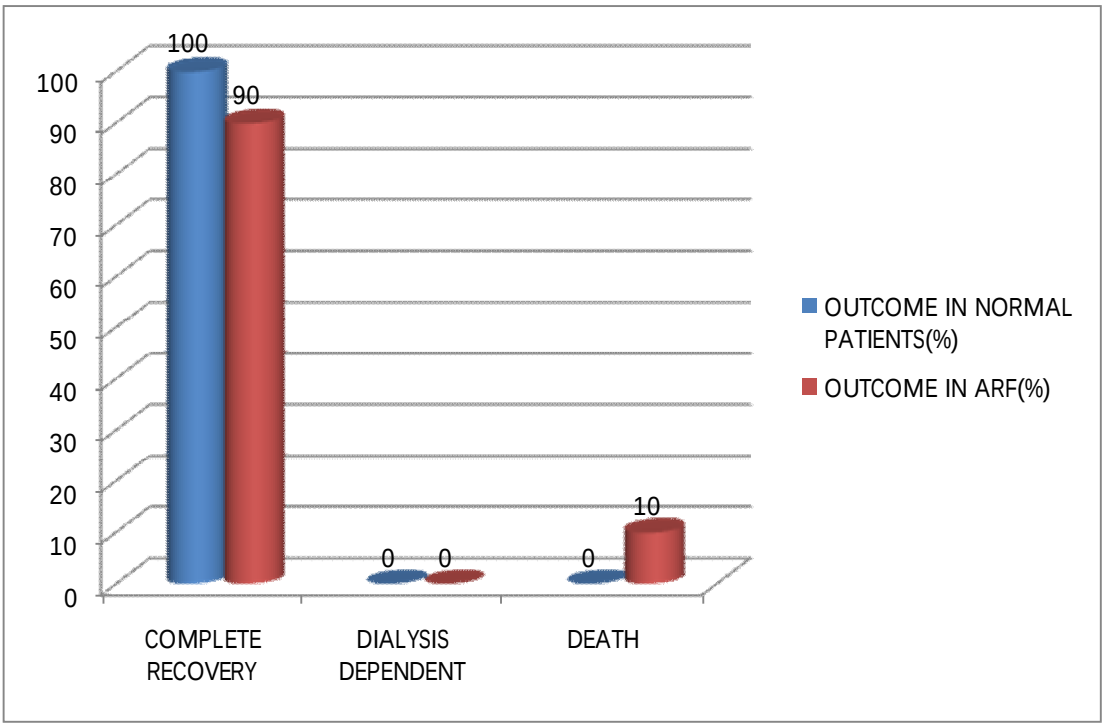


Out of 100 patients 5 needed hemodialysis.

TABLE 16: OUTCOME OF PATIENTS WITH SNAKE BITE

Outcome	Number	Percentage
Complete Recovery	98	98
Mortality	2	2

FIGURE 33: OUTCOME OF PATIENTS WITH SNAKE BITE



Out of 100 patients only 2 patients were went to mortality and remaining 98 patients recovered completely.

TABLE 17: LEVELS OF SERUM CREATININE

Creatinine values of Ist day and IInd day

	N	Range	Minimum	Maximum	Mean	Std. Deviation	p
CR Day1	100	1.6000	.6000	2.2000	.874000	.2866103	0.000
CR Day2	100	3.3000	.7000	4.0000	1.185000	.6484441	
Valid N (listwise)	100						

There exists statistical significance between I^{st} day and II^{nd} day creatinine values P value <0.0001.

TABLE 18: PLOTTING CREATININE LEVEL Vs OLIGUIRIA

CREATININE	OLIGUIRIA	
	Yes	**No**
Elevated	17 (85%)	0 (0%)
Not Elevated	3 (15%)	80(100%)
Total	20 (100%)	80(100%)

Sensitivity = 85%

Specificity = 100%

Positive Predictive Value = 100%

Negative Predictive Value = 93.69%

Diagnostic accuracy = 97%

Kappa agreement = 0.901

MC. Nemar (Paired test) P = 0.250

We have tested creatinine with respect to oliguria and found that 85% sensitivity, 100% specificity and diagnostic accuracy 97%. We also seen kappa measure of agreement between creatinine based renal failure and oliguria based renal failure and value is 0.901, which is almost agreement between two.

TABLE 19: PLOTTING RENAL FUNCTION Vs GENDER

Gender	Renal Failure	
	Yes	**No**
Male	15 (75%)	53 (66.3%)
Female	5 (25%)	27 (33.8%)
Total	20 (100%)	80 (100%)

Chi-square = 0.563

P Value = 0.4531

Odds ratio = 1.528

There is no statistical significant between renal failure and normal outcome patients with respect to gender. Among renal failure patients 75% were male. If a patient is a male, there is 1.528 times an odds than female to get oliguria.

TABLE 20: PLOTTING RENAL FUNCTION Vs TIME LAG

Time Lag	Renal Failure	
	Yes	**No**
>5 Hours	16 (80%)	27 (33.8%)
≤ 5 Hours	4 (20%)	53 (66.3%)
Total	20 (100%)	80 (100%)

Chi-square = 13.96

P Value < 0.0001

Odds ratio = 7.852

There exists a statistical significance between renal failure and normal patient with respect to time lag between > 5 Hrs (delayed) and ≤ 5 Hrs.

Patients who were brought to hospital > 5 hours for treatment have an odds of 7.852 times than others for getting renal failure.

TABLE 21: PLOTTING RENAL FUNCTION Vs WBCT

WBCT	Renal Failure	
	Yes	**No**
> 20 Minutes	19 (95%)	51 (63.8%)
≤ 20 Minutes	1 (5%)	29 (36.3%)
Total	20 (100%)	80 (100%)

Chi-square = 7.44

P Value = 0.006377

Odds ratio = 10.8

There exists a statistical significance between renal failure and normal patients with respect to WBCT > 20 and WBCT ≤ 20 Minutes.

The patients those who are having WBCT > 20 Minutes are having 10.8 odds than WBCT ≤ 20 Minutes for getting renal failure.

TABLE 22: PLOTTING RENAL FUNTION Vs HYPOTENSION

Blood Pressure	Renal Failure	
	Yes	**No**
Hypotension	14 (70%)	1 (1.3%)
Normal	6 (30%)	79 (98.8%)
Total	20 (100%)	80 (100%)

Chi-square = 59.31

P Value < 0.0001

Odds ratio = 184.3

There exists a statistical significance between renal failure and normal patients with respect to hypotension / Normal.

The patients those who are hypotensive are having 184.3 odds than normotensive to get renal failure.

TABLE 23: PLOTTING RENAL FUNCTION Vs CELLULITIS

CELLULITIS	Renal Failure	
	Yes	**No**
Present	19 (95%)	7 (8.8%)
Absent	1 (5%)	73 (91.2%)
Total	20 (100%)	80 (100%)

Chi-square = 61.86

P Value < 0.0001

Odds ratio = 198.1

There exists a statistical significance between renal failure and normal patients with respect to cellulitis.

The patients with cellulitis are having 198.1 odds than non – cellulitis patients to get renal failure.

TABLE 24: PLOTTING RENAL FUNCTION Vs HEMATURIA

Hematuria	Renal Failure	
	Yes	**No**
Present	18 (90%)	8 (10%)
Absent	2 (10%)	72 (90%)
Total	20 (100%)	80 (100%)

Chi-square = 53.22

P Value < 0.0001

Odds ratio = 81

There exists a statistical significance between renal failure and normal patients with respect to hematuria Present/Absent.

The patients with hematuria are having 81 odds than non-hematuria patients for getting renal failure.

DISCUSSION

1. AGE OF THE PATIENT

Table 25: Comparison of Age Group in Various Studies

Age in years	Nishioka[59]	Hati[10]	Present study
21-30	21.6%	38.4%	17%
31-40	17.5%	18.9%	28%
41-50	10.6%	12.1%	23%
51-60	7.5%	4.9%	14%
Above 60	2.4%	4.2%	8%

Study done by Nishioka et al[59] had (17.5%) and Hati et al[10] had (18.9%) of snake bite in the age group between 31-40 years but in my study (28%) of patients were in this age group.

GENDER OF THE PATIENT

Table 26: Comparison of Gender Incidence in Various Studies

Gender	Banerjee[79]	Sawai[78]	Present study
Male	75%	66%	68%
Female	25%	33%	32%

In the present study incidence of snake bite were male (68%) and female (32%) as comparable with Sawai et al[78].

Males were the victims for snake bites more often than females in all studies including the present study. Agricultural labour is carried out predominantly by males and hence they are at risk.

SITE OF SNAKE BITE

Table 27: Comparison of site of bite in various studies

Site	Banerjee[79]	Sawai[78]	Present study
Lower limb	62.4%	67.8%	64%
Upper limb	24.5%	25.2%	30%

Most of the snake bites were observed in lower limb, followed by upper limb in all the above mentioned studies including the present study.

TIME LAG IN HOSPITALIZATION

Table 28: Lapse of time > 5 Hours in presenting to the hospital

Studies	Percentage
Patil BT et al[84]	42%
Athappan G et al[80]	55%
Present study	55%

In the present study 55% patients reported to the hospital >5 hours as comparable with Athappan series (55%).

Early hospitalization below 5 hours was shown to reduce the incidence of morbidity & mortality and also decreased the duration of hospital stay.

LOCAL ENVENOMATION

Table 29: Comparison of symptoms with other studies

Symptoms	Athappan G et al[80]	Present study
Signs of inflammation	98.7%	99%

Signs of inflammation in the present study were 99% which is comparable with Athappan et al[80] . In the present study pain was noted in 99% of the cases, 81% cases showed swelling, similar to Mishra's series[38] (100%) and Sarangi's series[55] (84%).

WHOLE BLOOD CLOTTING TIME

Table 30: Comparison of WBCT > 20 Mins with other study

Studies	Present study
Paul J Dasgupta et al[83]	74%
Present study	70%

WBCT >20 Mins is comparable with the study of Paul J Dasgupta et al[83].

ACUTE RENAL FAILURE IN SNAKE BITE

Table 31: Comparison of ARF in various studies

	Srilatha et al[64]	Chugh et al[65]	Present study
ARF	34%	28.8%	20%

The incidence of ARF in srilatha et al[64] was 34% and chugh et al was 28.8% but in my study it is 20%.

SIGNIFICANT VARIABLES

Table 32: Variables with significant p-value for the development of Snake bite induced ARF

Variables	Chi-square	P Value	Odds ratio
Time lag >5 hours	13.96	<0.0001	7.852
WBCT >20Min	7.44	0.006377	10.8
Hypotension	59.31	<0.0001	184.3
Cellulitis	61.86	<0.0001	198.1
Hematuria	53.22	<0.0001	81

Clinical variable like lapse in time, WBCT >20 Mins, Cellulitis, hypotension & hematuria had statistical significance with development of ARF. In present study patients showed local envenomation (99%), hypotension (15%), hematuria (26%) and oliguria (20%).

Srilatha et al[64] found hematuria in 40% and oliguria 34% of patients with ARF. Mishra et al[25] study had 25% of cases developing hypotension.

CONCLUSION

- Snake bites are common in and around Vellore district.
- Majority of the patients had signs of envenomation and a small number had snake bite without systemic envenomation.
- Snake bite is an important and preventable cause of ARF.
- Causes for ARF in snake bite are “multifactorial” in origin. Bleeding and hypotension are the important causative factors.
- Type of snake bite is one of the important factors in the development of Acute Renal Failure. ARF is commonly associated with Viperidea species.
- Delay in time in presenting to the hospital is one of the valid predictors of poor outcome in snake bite induced Acute Renal Failure.
- Snake bite patients developing coagulopathy are more prone to develop ARF. Snake bite patients presented with cellulitis as local manifestation are at increased risk to develop ARF.
- In ARF complicated cases, Dialysis & supporting therapy appears to be the mainstay of treatment.
- Anuria of more than 48 hours, severe hyperkalemia resistant to Medical therapy, pulmonary oedema, and progressive rise in Blood urea and serum creatinine are the main indications for dialysis in ARF.
- Clinical outcome depends on optimum ASV dosage which is variable, depending on the type of snake, type of envenomation, organ system involved & supportive care offered.

BIBLIOGRAPHY

1. Aurebch SP, Norris LR.Disorders caused by Reptile Bites and Marine animal exposures. 18th ed.Chapter 396 In: Harrison's Principles of Internal Medicine.
2. Jena Indramani, SarangiAkulananda. Snakes of Medical Importance And Snake Bite Treatment. Publication House, New Delhi 1993: 230-242.
3. Sachdev. Principles Of Pediatric And Neonatal Emergencies 1ST Ed, 1990: 259- 266.
4. Fielding H. Garrison An Introduction To The History Of Medicine, 4TH Ed, 1929. 342-356.
5. Pillay Vv. Irritants Of Animal Origins, Chapter 3 In Medical Toxicology, Jaypee Brothers New Delhi, 2001:97-107.
6. OttenEj. Venomous Animal Injuries. In: Rosen P, Barkin R, Editors. Emergency Medicine Concepts And Clinical Practices. New York: Mosby; 1998. Pp. 924–40.
7. Sachdev. Principles Of Pediatric And Neonatal Emergencies 1ST Ed, 1990: 259- 266.
8. BawaskarHs. Snake Venoms AndAntivenoms: Critical Supply Issues. J Assoc Physicians India 2004; 52: 11-12.
9. Warell Da. Animal Poisons From Manson's Tropical Disease, 19TH Ed, Elbs 1987; 65: 855-882.

10. HatiAk, Mandal M, De Mk, Mukherjee H, HatiRn. Epidemiology Of Snake Bite In The District Of Burdwan, West Bengal. J Indian Med Assoc. 1992;90:145–7.
11. Gaitonde Bb, Bhattacharya S. An Epidemiological Survey Of Snake-Bite Cases In India. Snake. 1980;12:129–33.
12. Philip E. Snake Bite And Scorpion Sting. In: SrivatavaRn, Editor. Pediatric And Neonatal Emergency Care. New Delhi: Jaypee Brothers; 1994. Pp. 227–34.
13. Laxman S, Jambagi A. A Clinical Study Of Snake Bite In And Around Harangi, A Rural Karnataka, Ashirwad Nursing And Maternity Home, Kjms, Internet Ed: P-9.
14. DeorasPj. Snakes Of India; 4TH Ed (Revised), National Book Trust, India 1999: 65-70.
15. Banerjee Rn. Poisonous Snakes Of India, Their Venoms, Symptomatology And Treatment Of Envenomation. Progress In Clinical Medicine In India By M.M.S. Ahuja; Ii Ed. Arnold Heinmann Publishers; 2; 1983: 136-179.
16. Seneviratne U, Dissanayake S. Neurological Manifestations Of Snakebite In Sri Lanka. J Postgrad Med. 2002;48:275–8.
17. Warrell Da. Injuries, Envenoming, Poisoning, And Allergic Reactions Caused By Animal. In: Warrell Da, Cox Tn, Firth Jd,

Benj J Jr, Editors. Oxford Textbook Of Medicine. Oxford: Oxford University Press; 2003. Pp. 923–45.

18. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S. Snake Envenomation In A North Indian Hospital. Emerg Med J. 2005;22:118–20.

19. Theakston Rd, Warrell Da, Griffiths E. Report Of A Who Workshop On The Standardization And Control Of Antivenoms. Toxicon. 2003;41:541–57.

20. Parekh Ck. Text Book Of Forensic Medicine And Toxicology, Jaypee Brothers 2000; 9:41-4

21. Kulkarni Ml, Vijay Kumar Km. Snake Bite, Recent Advances In Paediatrics Tropical Paediatric, 4TH Ed, Jaypee Brothers, New Delhi 1991: 394 -410.

22. PradeepBambery, Snake Bite And Arthropod Envenomation, Api Textbook Of Medicine, 7TH Ed, Api Mumbai 2003: 1279 -1281.

23. Simpson Id, Norris Rl. Snakes Of Medical Importance In India: Is The Concept Of The “Big 4” Still Relevant And Useful? Wilderness Environ Med. 2007;18:2–9.

24. BhatRn. Viperine Snake Bite Poisoning In Jammu. J Indian Med Assoc. 1974;63:383–92.

25. SainiRk, Sharma S, Singh S, Pathania Ns. Snake Bite Poisoning: A Preliminary Report. J AssocPhys India. 1984;32:195–7.

26. Warrell Da. Who/Searo Guidelines For The Clinical Management Of Snakebite In The Southeast Asian Region. Se Asian J Trop Med Pub Hlth. 1999;30:1–85.
27. Reid Ha. Prolonged Defibrination Syndrome After Bite By Carpet Viper EchisCarinatus.Bmj 1977 June; 2: 123-25
28. SainiRk, AryaRk, Singh S, Sharma S. Coagulation Defects In Snake Bite Poisoning. J Assoc Physicians India 1985 Jan; 33: 12-15.
29. John. B. Harries Snake Venoms In Science And Clinical Medicine. Neuropharmacological Aspects Of The Activity Of Snake Venoms Trans Royal Soc Trop Med Hyg 1989; 83: 745-747.
30. Muzumdar G, Basu J, GhoshRn, GhoshJc, Chettri Mk. Cardiac Profile In Viper Venom Poisoning. Indian Heart Journal 1982; 34: 283.
31. Joshua Wyhne, Eugene Braunwald. Cardiac Complications Of Snake Bite. Braunwald Heart Disease. A Text Book Of Cardiovascular Medicine 7TH Ed, W. B. Saunders Company 1992: 1453-56.
32. RatcliffePj. Direct Nephrotoxicity Of Russell's Viper Venom Demonstrated In The Isolated Perfused Rat Kidney. Am J Trop Med Hyg 1989; 140 (3): 312 -319.

33. HagwoodBj. Sugh Alistair Reid ObeMd; Investigation And Treatment Of Snake Bite. Toxicon 1998; 36(3): 413-446.
34. Ramakrishnan Mk, Sankaran K, Gupta Gd, Chandrashekar's External OphthalmoplegiaIn Elapidae Bites And Its Response To Neostigmine. Neurology (India); 1975; 23; 105-110.
35. MyintLwin, Warrel Da, Phillips Re, Tin-Nu-Swe, Tun-Pe Et Al. Bites By Russell's Viper In Burma: Hemostatic, Vascular And Renal Disturbance And Response To Treatment. Lancet 1985 Dec 7; 2(8467): 1259-64.
36. SainiRk, AryaRk, Singh Sharma S. Coagulation Defects In Snake Bite Poisoning; J Assoc Physician India 1985 Nov; (5): 33-35.
37. Mitrakul C, Juzi U, Pongrujikorn W. Antivenom Therapy InRussel's Viper Bite, Ajcp 1991 March: 412-417.
38. Mishra Sn, NayakKc, Jain Ak. Profile Of Cardiac Complications Of Snake Bite. Indian Heart Journal 1990 May; 42 (3):16-20.
39. Reid Ha. Animal Poisons. Manson's Tropical Diseases, 18TH Ed, London. Balliere-Tindall Co 1982; 544-546.
40. Russell Fe. Snake Venom Poisoning. Philadelphia Jb Lippincott Company. 1980: 235-285.
41. Thein Than, Tin Tun Et Al. Development Of Renal Function Abnormalities Following Bites ByRusell's Viper In Myanmar. Trans Royal Soc Med Hyg 1991; 85: 404-409

42. TunPe, Fin Nu Warrell E. The Efficacy Of Tourniquets As A First Aid Measure For Russell's Viper Bite In Burma. Trans Royal Soc Trop Med Hyg.1987; 81: 403-405.
43. Reid Ha, ThekastonRdg. The Management Of Snake Bite. Bull Who 1983; 63: 885-895.
44. Nigam. Snake Bite: A Clinical Study. Ind J Trop Med Sc 1973; 27: 697.
45. Hasiba U, Rosenback Lm, Rockwell D. Dic-Like Syndrome After Envenomation By The Snake, CrotalusHorridusHorridus. N Eng J Med 1975; 292: 505 -7.
46. BudzynskiAz, PandyaBv, Rubin Rn. Fibrinogenolytic Afibrinogenaemia After Envenomation By Western Diamond Back Rattle Snake (Crotalusatrox). Blood 1984; 63: 1-14.
47. Weiss Hj, Phillips L, HopwellWs. Heparin Therapy In A Patient Bitten By A Saw-Scaled (EchisCarinatus), A Snake Whose Venom Activates Prothrombin. Am J Med 1973; 54: 653-62.
48. Chugh Ks, Pal Y, ChakravartyRn, DattaBn, Mehta R, Et Al. Acute Renal Failure Following Poisonous Snake Bite. Am J Kidney Dis 1984; 4: 30-8.
49. ShastryJc, Date A, Carman Rh, JohnyKv. Renal Failure Following Snake Bite. Am J Trop Med Hyg 1977; 26: 1032-8.

50. Mittal Bv, KinareSg, AcharyaVn. Renal Lesions Following Viper Bites-A Study Of 14 Years. Indian J Med Res 1986; 83: 642-51.
51. Mathew Mt, Rajaratnam K. Renal Papillary Necrosis InOphitoxemia (Abstract). J Assoc Physicians India 1987 April; 35: 20.
52. Efrati P, Reif L. Clinical And Pathological Observations On 65 Cases Of Viper Bite In Israel. Am J Trop Med Hyg 1953; 2:1085-108.
53. BurdmannEa, Woronik V, Prado Eb. Snakebite-Induced Acute Renal Failure: An Experimental Model. Am J Trop Med Hyg 1993; 48(1): 82-8
54. Chugh Ks, Sakhuja V. Renal Disease Caused By Snake Venom. Handbook Of Natural Toxins Tu At 3 Ed, New York Marcel Dekker Inc 1991; 471-93.
55. Sarangi A, Jena I, Sahoo H And Jp Das, Profile Of Snake Bite Poisoning With Special Reference To Hematological, Renal, Neurological And Electrocardiographic Abnormalities. J Assoc Physicians India 1977 Aug; 25: 654-63.
56. David A Warrell, Snake Venoms In Science And Clinical Medicine, Russell's Viper Biology Venom And Treatment Of Bites .Trans. Royal Society Of Trop. Med. And Hygiene.1989; 83: 732-740.

57. Tin Nu Swe, Warrell Da. Renal Ischemia Transient Glomerular Leak And Acute Renal Tubular Damage In Patients' Envenomated By Russell's Viper In Myanmar; Trans. Royal Society Of Tropical Medicine And Hygiene. 1993; 87: 678-681.
58. Reid Ha. Prolonged Defibrination Syndrome After Bite By Carpet Viper EchisCarinatus.Bmj 1977 June; 2: 123-25.
59. SilveriaPv, Nishioka C De A. Non Venomous Snake Bite And Snake Bite With Out Envenoming In A Brazilian Teaching Hospital. Analysis Of 91 Cases. Rev Inst Med Trop Sao Paulo 1992; 34(6): 499-503.
60. Merchant Mk, KhannaUb, Mittal Bv, Almeida Af, AcharyaVn. Clinicopathological Study Of Acute Renal Failure Following Viperine Snake Bite. J Assoc Physician India 1989 July; 37(7): 430-3.
61. Ho M, WarrellMj, Warrell Da, Bidwell D, Voller A. A Critical Reappraisal Of The Use Of Enzyme-Linked Immunosorbent Assays In The Study Of Snakebite. Toxicon. 1986;24:211–21.
62. Reprint Of The 1999 Edition Written And Edited For Seameotropmed – Regional Centre For Tropical Medicine, Faculty Of Tropical Medicine, MahidolUniversity, Thailand Guidelines For The Clinical Managementof Snake Bite In The South-East Asia Region,Pg-19

63. HawgoodBj, Hugh Ar. Investigation And Treatment Of Snakebite. Toxicon. 1998;36:431–4

64. Srilatha V, Ranganath J Al, Ravi Shankar S, Prasad G, Ravi Raju T. Acute Renal Failure In Patients With Venomous Snakebite. DeptOf Nephrology Andra Medical College, Visakhapatnam, India. Indian J Trop Medicine.1997; 9:456-67

65. Chugh KS. Snake bite induced acute Renal failure in India. Post graduate Institute of Medical Education and Research, Chandigarh, India. Kidney Int 1989 Mar, 35 (3) 891-907.

66. Warrell Da. Who/Searo Guidelines For The Clinical Management Of Snakebite In The Southeast Asian Region. Se Asian J Trop Med Pub Hlth. 1999;30:1–85.

67. NayakKc, Jain Ak, ShardaDp, Mishra Sn. Profile Of Cardiac Complications Of Snake Bite. Indian Heart J. 1990;42:185–8.

68. Theakston Rd, Warrell Da, Griffiths E. Report Of A Who Workshop On The Standardization And Control Of Antivenoms. Toxicon. 2003;41:541–57.

69. Norris Rl, Ngo J, Nohan K, Hooker G. Physicians And Lay People Are Unable To Apply Pressure Immobilisation Properly In A Simulated Snakebite Scenario. Wilderness Environ Med 2005;16:16–21.

70. HowarthDm, Southee As, WhytwIm. Lymphatic Flow Rates And First Aid In Simulated Peripheral Snake Or Spider Envenomation. Med J Australia. 1994;161:695–700.
71. Who. Regional Office For South-East Asia. The Clinical Management Of Snake Bites In The Southeast Region Asia. 2005
72. Warrell Da. Injuries, Envenoming, Poisoning, And Allergic Reactions Caused By Animal. In: Warrell Da, Cox Tn, Firth Jd, Benj J Jr, Editors. Oxford Textbook Of Medicine. Oxford: Oxford University Press; 2003. Pp. 923–45.
73. Clark Rf, MckinneyPe, Chase Pb, Walter Fg. Immediate And Delayed Allergic Reactions To Crotalidae Polyvalent Immune Fab (Ovine) Antivenom. Ann Emerg Med. 2002;39:671–6.
74. Dillon, Blackman. Venomous Snake Bite, Past, Present And Future Treatment Options. Journal Of American Board Of Family Practioner1992; (4): 399.
75. Sutherland Sk, Management Of Venomous Bite And Stings. Medicine International 3RD Ed, Middle Eastern Publication.1984: 1324-36.
76. Thomas Pp, Jacob J. Randomised Trial OfAntivenom In Snake Envenomation With Prolonged Clotting Time. Br Med J. 1985;291:177–8.

77. Tun P, Khin Ac. Amount Of Venom Injected By Russells Viper (ViperaRusselli). Toxicon. 1986;24:730–3.
78. Sawai Y. Study On Deaths Due To Snakebite In Anuradhapura District, Sri Lanka. Snake. 1984;16:7–15.
79. Banerjee Rn. Poisonous Snakes Of India, Their Venoms, Symptomatology And Treatment Of Envenomation. Progress In Clinical Medicine In India By M.M.S. Ahuja; Ii Ed. Arnold Heinmann Publishers; 2; 1983: 136-179.
80. Attappan G, Balaji MV, Navaneethan U, Thirumalaikolundu Subramanian P. Acute Renal Failure in Snake Envenomation: A Large Prospective Study. Saudi J Kidney Dis Transpl 2008: 19:404-10.
81. Mittal BV. Acute renal failure following poisonous snake bite. J Postgrad Med 1994:40:123.
82. Muthusethupathi MA. Acute Renal Failure due to snake bite. The Antiseptic 91(3):88-90.
83. Paul J, DasguptaS.Early Predictors of Acute Kidney Injury by Clinical Features of Snake bite Patients at the time of Hospital Admission. North Am J MED Sci 2012;4:216-20.
84. Patil BT. Snake Bite Induced Acute Renal Failure: A study of Clinical Profile and Predictors of Poor Outcome. 2012 Jun: 1 (2-3):59-65.

PROFORMA

PATIENT PARTICULARS

Name	:		Hospital	:
Age	:		I.P. No.	:
Sex	:	M/F	DOA	:
Occupation	:		DOD	:
Address	:			

EVIDENCE TO SNAKE BITE

1. Snake seen : Y/N
2. Fang marks seen : Y/N
3. Features of envenomation : Y/N

DETAILS OF SNAKE

• Snake: Seen /not seen /brought

• Species of snake if known:

DETAILS OF SNAKE BITE

• Site of bite : Lower limb : Right /left

Upper limb : Right /left

Other site:

• Presence of fang marks : Single/double

• Time of bite :

• Time of hospital admission :

• Interval between bite and medical aid :

• Premises : Indoor/outdoor

• Tourniquet : applied or not

CLINICAL EXAMINATION

General examination : Conscious/drowsy/stupor/coma

Vitals : Pulse : JVP: Elevated/Not

BP : mm/Hg

RR : /min

Temp : F

Pallor : Y/N

Icterus : Y/N

Cyanosis : Y/N

Clubbing : Y/N

Lymphadenopathy :Y/N

SIGNS OF ENVENOMATION

LOCAL EXAMINATION:

• Fang marks : Y/N

• Pain : Y/N

• Swelling : Y/N

• Cellulites : Y/N

• Bleeding : Y/N Others:

HAEMATOXIC FEATURES

• Bleeding from bite site : Y/N

• Bleeding from gums /gingival : Y/N

• Epistaxis : Y/N

• Ecchymosis / purpura /petechiae : Y/N

• Hematuria : Y/N

• Hematemesis : Y/N

• Hemoptysis : Y/N

NEUROTOXIC FEATURES

• Ptosis : Y/N

• Ophthalmoplegia : Y/N

• Pharyngeal paralysis : Y/N

• Respiratory paralysis : Y/N

• Flaccid limb paralysis : Y/N

SYSTEMIC EXAMINATION

CNS

• State consciousness : conscious/ unconscious Pupil: mm

• Speech :

• Cranial nerves :

• Motor system :

- **CVS**
- **RS**
- **KIDNEY** Normal
  Oliguria/Anuria

INVESTIGATIONS

• Complete haemogram : Hb TC DC ESR
Platelet

• Bleeding time :

• Whole blood clotting time :

• Urine examination : Albumin
Sugar
RBC

• Blood urea: Day 1: Day 2: Day 3: Day 4: Day 5:

• Serum creatinine: Day 1: Day 2: Day 3: Day 4: Day 5:

• Serum electrolytes:

• If any other investigation:

IMPRESSION:

Venomous /non venomous

Hemotoxic / Neurotoxic / Cardio toxic

TREATMENT

Anti-snake venom :

Total dose :

Hospital Stay :

Any adverse reactions : Y/N

Fasciotomy : Y/N site :

Need of organ support : Ventilator : No. of days

Dialysis : Y/N No of Sittings

OUTCOME

Complete recovery / Disability (morbidity) / Death (mortality):

MASTER CHART

S.No	Name	Age	Gender	Occup	ES	EMS	ENVENOM	SNAKE SEEN	SNAKE TYPE	SOB	FM	INTERVAL	PREMISES	TOURNIQUET	PULSE	BP SYSTOLE	BP DIASTOLE	RR	FM	PAIN	SWELL	CELLULITIS	BLEEDING	H1	H2	H3	H4	H5	H6	H7
1	INDIRA RANI	35	2		1	1	1	3	1	1	2	13	1	1	100	82	64	22	1	1	1	2	1	1	1	2	2	1	1	1
2	PAZHANI	48	1		1	2	2	2	0	1	2	7	1	1	108	90	60	20	1	1	1	1	1	1	1	2	2	1	2	2
3	NITHYA	18	2		1	1	2	1	0	1	1	8	2	2	94	120	70	18	1	1	1	2	2	2	2	2	2	2	2	2
4	MANNU	35	2		1	1	2	1	0	1	2	5	1	1	96	100	60	17	1	1	1	2	1	1	2	2	2	2	2	2
5	AIYAPPAN	30	1		1	1	1	1	3	1	2	24	1	1	84	110	70	20	1	1	2	2	1	2	2	2	2	2	2	2
6	INDRA	40	2		1	1	2	3	1	1	2	7	1	1	86	110	70	18	1	1	1	1	1	2	2	2	2	1	2	1
7	THILAGA	32	2		1	2	2	1	0	1	2	25	1	2	96	100	60	18	1	1	1	2	1	2	2	2	2	2	2	2
8	INDIRA KUMARI	57	2		2	1	2	2	0	2	2	2	1	2	89	100	80	19	1	1	1	1	1	2	2	2	2	2	2	2
9	MANOHARAN	60	1		1	1	2	1	1	1	2	12	2	2	100	130	80	19	1	1	1	2	2	2	2	2	2	2	2	2
10	MOHANA	26	2		1	1	2	1	0	2	2	5	1	1	100	120	80	20	1	1	1	2	1	2	2	2	2	2	2	2
11	JAGANATHAN	40	1		1	1	2	1	0	1	2	2	1	1	98	130	70	21	1	1	2	2	2	2	2	2	2	2	2	2
12	PARASURAM	42	1		1	1	2	1	0	2	2	3	1	1	108	120	80	18	1	1	1	2	1	2	2	2	2	2	2	2
13	BALAJI	45	1		1	1	2	1	0	1	2	2	1	1	100	120	70	18	1	1	1	2	1	2	2	2	2	2	2	2
14	UNNAMALAI	75	2		1	1	2	1	0	1	2	6	1	1	91	140	80	19	1	1	1	2	1	2	2	2	2	2	2	2
15	SAVITHRI	40	2		1	1	1	1	1	2	2	40	1	2	72	120	80	14	1	1	1	1	1	1	2	2	2	2	2	2
16	SELVARAJ	46	1		1	1	2	1	0	2	1	4	1	1	82	120	80	16	1	1	1	2	1	1	2	2	2	2	2	2
17	PARVATHY	45	2		1	1	2	1	1	2	2	4	1	1	76	110	70	14	1	1	1	2	2	2	2	2	2	2	2	2
18	ARJUNAN	47	1		1	1	2	1	0	1	1	3	1	1	76	110	70	16	1	1	1	2	1	2	2	2	2	2	2	2
19	ARUMUGAM	56	1		1	1	2	1	0	1	2	2	1	1	72	120	80	16	1	1	1	2	1	1	2	2	2	2	1	2
20	MANJULA	35	2		1	1	2	1	1	1	1	3	1	2	80	130	70	15	1	2	2	2	1	1	1	2	2	2	2	1
21	GOPAL	85	1		2	1	2	2	1	2	2	2	2	1	76	140	90	18	1	1	1	2	1	1	1	1	2	2	2	2
22	VIJAYAN	13	1		2	1	1	2	0	1	2	13	2	2	82	100	70	16	1	1	1	2	1	1	2	1	1	2	2	2
23	TAMILARASI	40	2		1	1	2	1	2	1	2	4	2	1	72	110	60	14	1	1	2	2	1	1	2	2	2	2	2	2
24	SUBRAMANI	65	1		1	1	1	1	1	3	2	5	1	2	76	110	70	16	1	1	2	2	2	2	2	2	2	2	2	2
25	RANGANATHAN	60	1		1	1	2	1	0	3	1	2	1	2	74	120	80	16	1	1	1	2	1	1	1	2	2	2	2	2
26	KUMAR	45	1		1	1	2	1	0	1	2	3	1	1	72	120	80	18	1	1	1	2	2	2	2	2	2	2	2	2
27	MURUGAN	66	1		1	2	2	1	0	1	2	5	1	2	108	90	60	20	1	1	1	1	1	2	2	2	2	2	2	2
28	PICHANDI	22	1		1	1	1	1	1	2	2	21	1	1	96	90	60	20	1	1	1	1	1	1	2	1	2	2	2	2
29	GOWRI	35	2		1	1	1	1	0	2	2	11	1	1	68	130	60	20	1	1	1	2	2	2	2	2	2	2	2	2
30	PERUMAL	35	1		1	1	2	1	1	1	2	5	1	1	90	110	70	18	1	1	1	1	1	1	1	2	2	2	1	1
31	THANJAMMAL	46	2		1	1	2	1	0	2	1	4	1	1	96	90	60	19	1	1	1	2	1	1	1	1	2	2	2	2

32	SUMATHI	28	2		1	2	2	1	1	2	0	2	1	2	98	100	60	18	2	1	2	2	1	1	2	2	2	2	2	2
33	RAJ KUMAR	15	1		1	1	2	1	0	1	1	3	1	1	87	100	60	19	1	1	1	2	2	1	2	2	2	2	2	1
34	JAYANTHI	38	2		1	2	2	1	1	1	0	3	1	1	94	110	80	16	2	1	1	2	2	2	1	2	2	2	2	1
35	ANANDHAN	40	1		1	1	1	1	2	2	2	5	1	1	106	100	60	20	1	1	2	2	2	2	2	2	2	2	2	2
36	VIVEK	27	1		1	1	2	3	1	1	2	3	1	1	84	110	80	19	1	1	1	1	1	2	2	2	2	2	2	2
37	PARAMESWARAN	40	1		1	1	1	1	2	3	2	3	2	2	100	140	80	20	1	1	1	2	2	2	2	2	2	2	2	2
38	TAMILSELVI	40	2		1	1	2	1	2	3	2	2	2	2	110	140	90	21	1	1	2	2	2	2	2	2	2	2	2	2
39	SETTU	41	1		1	1	2	1	0	1	2	5	1	1	89	110	80	16	1	1	1	2	2	2	2	2	2	2	2	2
40	SHANTHI	45	2		1	1	1	1	1	2	2	6	1	1	98	100	80	19	1	1	1	2	2	1	2	2	2	2	2	2
41	VENKATESAN	30	1		1	1	2	1	1	1	2	4	1	1	98	120	70	16	1	1	1	2	2	1	1	2	2	2	2	2
42	CHINNAPAYAN	47	1		1	1	2	1	0	1	1	25	1	1	70	110	80	15	1	1	1	2	2	1	1	1	2	2	2	2
43	VINAYAGAM	25	1		1	1	2	1	0	3	2	6	1	2	76	120	80	17	1	1	2	2	1	1	2	2	2	2	2	2
44	MANI	40	1		1	1	2	1	0	1	2	4	1	1	72	130	70	13	1	1	1	2	1	1	2	2	2	2	1	2
45	VELLIKANNI	50	2		1	1	1	1	0	2	1	6	1	1	74	110	80	14	1	1	1	2	1	1	1	2	2	2	2	2
46	KANNIYAPPAN	33	1		1	1	2	1	0	1	2	2	1	1	82	120	80	16	1	1	2	2	2	2	2	2	2	2	2	2
47	CHINNAPAYAN	47	1		1	1	2	1	1	2	2	1	1	1	72	110	70	16	1	1	1	2	2	1	2	2	2	2	2	2
48	VELAN	40	1		1	1	1	1	0	1	1	6	1	1	90	100	60	18	1	1	2	2	2	2	2	2	2	2	2	2
49	RADHA	25	2		1	1	1	1	1	1	2	18	1	1	100	90	60	19	1	1	1	2	1	1	1	2	2	2	2	2
50	RANI	28	2		1	1	1	1	0	1	2	15	1	1	80	100	60	18	1	1	1	1	1	2	2	2	2	2	2	2
51	MARI	34	1		1	1	2	1	1	1	2	4	1	1	90	120	60	16	1	1	1	2	1	2	2	2	2	2	2	2
52	GOPI	26	1		1	1	2	1	0	2	1	7	1	1	68	120	80	18	1	1	1	2	1	1	1	2	2	1	2	1
53	SAMBASIVAM	50	1		1	1	2	1	0	2	2	8	1	1	81	120	80	18	1	1	1	2	1	1	1	2	2	2	2	2
54	SUBRAMANI	68	1		1	1	1	1	1	1	1	24	1	1	96	120	80	21	1	1	1	1	1	1	1	1	2	1	2	2
55	MANIVASAGAM	59	1		1	1	2	1	0	1	2	7	1	1	90	120	80	19	1	1	2	2	2	1	2	2	2	2	2	2
56	SRINIVASAN	32	1		1	1	2	3	1	1	2	2	1	1	102	120	80	20	1	1	2	2	2	2	2	2	2	2	2	2
57	THANJAMMAL	45	2		1	1	2	1	0	1	2	2	1	1	90	120	80	14	1	1	1	2	1	1	2	2	2	2	2	2
58	KAKKAN	18	1		1	1	1	1	0	2	2	3	1	2	80	120	80	16	1	1	1	2	2	2	2	2	2	2	2	2
59	KRISHNAN	55	1		1	1	2	1	0	2	2	3	1	1	80	120	80	18	1	1	1	2	2	2	2	2	2	2	2	2
60	PONNUSAMY	50	1		1	1	1	1	0	2	2	28	1	1	76	110	80	18	1	1	1	1	1	1	1	2	2	1	2	1
61	MUNIRATHNAM	53	1		1	1	2	1	1	1	2	8	1	2	74	100	60	12	1	1	1	2	2	1	2	2	2	1	1	2
62	SANGEETHA	25	2		1	1	2	1	0	1	1	3	1	1	78	100	70	14	1	1	1	2	2	1	2	2	2	2	2	2
63	KANNIYAPPAN	18	1		1	1	2	1	0	1	2	5	1	1	70	110	70	16	1	1	1	2	1	1	1	2	2	2	2	2
64	SRINIVASAN	21	1		1	1	2	1	0	1	2	4	1	2	82	110	70	14	1	1	1	2	2	1	1	2	2	2	2	1
65	JAYAPAL	38	1		1	1	2	1	0	1	2	2	1	2	80	130	90	16	1	1	1	2	2	2	2	2	2	2	2	2
66	TAJUDEEN	18	1		1	1	1	1	0	1	2	7	1	1	84	100	80	14	1	1	1	1	2	2	2	2	2	2	2	2
67	PARTHIBAN	22	1		1	1	1	1	2	1	2	4	2	1	108	120	90	20	1	1	2	2	2	2	2	2	2	2	2	2
68	KANIMOZHI	32	2		1	1	2	1	0	2	2	2	1	2	88	120	90	17	1	1	1	2	2	1	2	2	2	1	2	2
69	AMUDHA	38	2		1	1	1	1	2	2	2	3	1	1	90	130	80	16	1	1	2	2	2	2	2	2	2	2	2	2
70	NARASIMMA NAIDU	66	1		1	1	2	1	0	1	2	5	1	1	76	130	80	16	1	1	1	2	1	1	1	2	2	2	2	2

71	PARTHIBAN	20	1		1	1	1	1	0	2	2	2	1	1	70	110	70	16	1	1	2	2	2	1	2	2	2	1	1	2
72	CHINNA KRISHNAN	39	1		1	1	2	1	0	2	2	4	1	1	78	110	80	16	1	1	1	2	2	1	2	2	2	2	2	1
73	JAYAKUMAR	57	1		1	1	1	1	0	1	2	24	1	1	89	120	80	19	1	1	1	1	1	1	1	2	2	1	2	1
74	VIJAYALAKSHMI	30	2		1	1	2	1	0	1	2	2	1	1	96	100	60	18	1	1	2	2	2	2	2	2	2	2	2	2
75	CHINNAPPA	68	1		1	1	1	1	2	2	2	17	1	1	60	100	60	20	1	1	2	2	2	2	2	2	2	2	2	2
76	SHANKAR	45	1		1	1	2	1	0	3	2	2	2	2	79	100	80	15	1	1	1	2	2	1	2	2	2	1	2	2
77	SABARINATHAN	32	1		1	1	2	3	1	1	2	5	1	1	96	120	90	19	1	1	1	2	1	2	2	2	2	2	2	2
78	ALAGESAN	35	1		1	1	1	1	0	2	2	2	1	1	96	130	70	24	1	1	2	2	2	2	2	2	2	2	2	2
79	PADMAVATHY	47	2		1	1	1	1	1	2	2	5	1	1	93	130	80	20	1	1	1	2	2	1	1	2	2	2	1	2
80	PALANI	48	1		1	1	1	1	1	1	2	7	1	1	80	90	60	14	1	1	1	2	1	1	1	2	1	1	2	2
81	CHINNAKOLANDAI	60	2		1	1	1	1	1	1	2	20	1	1	104	80	60	21	1	1	1	1	1	1	1	1	2	1	2	1
82	MURUGAPPAN	51	1		1	1	1	1	0	1	2	10	1	2	108	70	50	18	1	1	1	2	1	1	1	1	2	1	2	2
83	VENKATESAN	56	1		1	1	2	1	0	1	2	2	1	1	89	86	60	18	1	1	1	2	2	1	1	2	2	2	2	2
84	PASUPATHY	19	1		1	1	1	1	1	1	2	10	1	1	104	80	50	21	1	1	1	1	1	1	1	1	2	1	2	1
85	ELUMALAI	50	1		1	1	1	1	1	1	2	90	1	1	110	80	50	26	1	1	1	2	2	1	1	1	2	2	2	2
86	SUBURAYAN	47	1		1	1	2	1	0	1	2	2	1	1	84	82	60	18	1	1	1	2	2	1	1	2	2	2	2	2
87	DAYALAN	45	1		1	1	1	3	1	1	2	9	1	1	104	80	60	20	1	1	1	1	2	1	2	2	2	1	2	1
88	SUMATHI	35	2		1	1	1	1	0	2	2	8	1	1	80	80	50	27	1	1	1	1	2	1	2	2	2	1	2	1
89	PARTHASARATHY	18	1		1	1	1	1	0	1	2	4	1	1	106	70	50	26	1	1	1	1	1	1	2	2	2	1	2	2
90	HEMAVATHY	20	2		1	1	1	1	0	1	2	8	2	1	89	100	60	14	1	1	1	1	1	1	1	2	2	1	2	2
91	MAYAKRISHNAN	40	1		1	1	1	1	0	1	2	24	1	1	94	120	90	18	1	1	1	1	1	1	2	2	2	2	1	2
92	SARIDHA	30	2		1	1	1	1	0	1	2	8	1	2	81	110	70	15	1	1	1	1	1	1	1	2	2	2	2	2
93	CHANDRA	60	2		1	1	1	1	0	2	2	12	1	1	98	70	50	21	1	1	1	1	1	1	1	2	2	1	1	2
94	NANDHAKUMAR	30	1		1	1	1	1	1	1	2	10	2	1	114	70	40	20	1	1	1	1	1	1	2	2	2	1	2	2
95	CHAKKARAI	65	1		1	1	2	1	0	1	2	3	1	1	89	100	60	20	1	1	1	2	2	2	1	2	2	1	2	2
96	JANAKIRAMAN	48	1		1	1	2	1	0	2	2	6	1	1	92	100	60	20	1	1	1	1	1	1	2	2	2	1	1	2
97	GUNASEKAR	40	1		1	1	1	1	0	1	2	24	1	1	120	60	40	26	1	1	1	1	1	1	1	2	2	1	1	2
98	MUNUSAMY	60	1		1	1	1	1	0	1	2	5	1	1	90	60	40	28	1	1	1	2	1	1	2	2	2	1	2	1
99	MANIKANDAN	29	1		1	1	2	1	0	1	1	1	1	1	91	100	70	20	1	1	1	2	2	2	2	2	2	2	2	2
100	NATRAJAN	55	1		1	1	1	1	0	1	2	12	1	1	112	70	50	22	1	1	1	1	1	1	1	2	2	1	2	2

MASTER CHART

S.No	N1	N2	N3	N4	N5	RENALFUNCTION	PLATELETS	WBCT	U1	U2	U3	UR1	UR2	UR3	UR4	UR5	CR1	CR2	CR3	CR4	CR5	ASV	STAY IN WARDS	ADVERSE	FASCIOTOMY	VENT SUPPORT	DIALYSIS	SITTINGS	OUTCOME
1	2	2	2	2	2	2	25000	2	2	2	1	60	71	70	50	42	1.5	3.2	3	2.1	1.1	30	7	2	2	2	1	3	1
2	1	1	2	2	2	1	80000	2	2	2	2	48	54	50	46	0	1.1	1.5	1.5	1.1	0	26	6	2	2	2	2	0	1
3	2	2	2	2	2	1	2E+05	1	2	2	2	24	25	0	0	0	0.7	0.8	0	0	0	10	3	2	2	2	2	0	1
4	2	2	2	2	2	1	1E+05	2	2	2	2	28	30	0	0	0	0.8	0	0	0	0	18	4	2	2	2	2	0	1
5	1	1	2	2	2	1	1E+05	1	2	2	2	20	22	0	0	0	0.7	0.8	0	0	0	10	3	2	2	2	2	0	1
6	1	1	2	2	2	1	90000	2	2	2	1	32	34	0	0	0	0.9	0.9	0	0	0	20	4	2	2	2	2	0	1
7	2	2	2	2	2	1	1E+05	2	2	2	2	28	0	0	30	0	0.7	0	0	0.7	0	20	3	2	2	2	2	0	1
8	2	2	2	2	2	1	90000	2	2	2	2	26	0	27	0	0	0.7	0	1	0	0	15	6	2	2	2	2	0	1
9	1	1	2	2	2	1	2E+05	1	2	2	2	28	0	30	0	0	0.9	0	0.9	0	0	10	4	2	2	2	2	0	1
10	1	1	2	2	2	1	86000	1	2	2	2	35	0	33	0	35	1	0	1	0	1	13	7	2	1	2	2	0	1
11	2	2	2	2	2	1	1E+05	1	2	2	2	26	0	26	0	0	0.7	0	0.7	0	0	10	4	2	2	2	2	0	1
12	1	1	2	2	2	1	1E+05	1	2	2	2	37	0	37	0	0	0.9	0	1	0	0	10	4	2	2	2	2	0	1
13	2	2	2	2	2	1	2E+05	2	2	2	2	25	0	30	0	0	0.8	0	0.8	0	0	18	4	2	2	2	2	0	1
14	2	2	2	2	2	1	1E+05	1	2	2	2	30	0	40	0	33	0.6	0	0.9	0	0.8	20	5	2	2	2	2	0	1
15	1	1	2	2	2	1	2E+05	1	2	2	2	25	0	0	30	0	0.7	0	0	0.7	0	8	5	2	2	2	2	0	1
16	1	1	2	2	2	1	1E+05	2	2	2	2	30	0	30	0	0	0.8	0	0.8	0	0	8	4	2	2	2	2	0	1
17	2	2	2	2	2	1	4E+05	1	2	2	2	24	48	0	0	0	0.8	1.2	0	0	0	20	4	2	2	2	2	0	1
18	1	1	2	2	2	1	2E+05	1	2	2	2	20	40	28	0	0	0.5	1.3	0.8	0	0	15	6	2	2	2	2	0	1
19	2	2	2	2	2	1	1E+05	2	2	2	2	30	0	31	0	0	0.9	0	1	0	0	10	4	2	2	2	2	0	1
20	1	1	2	2	2	1	2E+05	1	2	2	2	40	0	0	0	0	1	0	0	0	0	10	6	2	2	2	2	0	1
21	2	2	2	2	2	1	37000	2	2	2	2	19	0	0	23	0	0.6	0	0	0.8	0	20	5	2	2	2	2	0	1
22	2	2	2	2	2	1	1E+05	2	2	2	2	28	0	28	0	0	0.7	0	0.6	0	0	10	4	2	2	2	2	0	1
23	1	1	2	2	2	1	3E+05	1	2	2	2	16	0	0	0	0	0.7	0	0	0	0	20	3	2	2	2	2	0	1
24	2	2	2	2	2	1	1E+05	2	2	2	2	24	0	26	0	0	0.6	0	0.8	0	0	10	4	2	2	2	2	0	1
25	2	2	2	2	2	1	1E+05	2	2	2	2	22	24	0	0	0	0.6	0.9	0	0	0	23	7	2	2	2	2	0	1
26	2	2	2	2	2	1	3E+05	1	2	2	2	36	0	0	0	0	0.9	0	0	0	0	5	3	2	2	2	2	0	1
27	2	2	2	2	2	1	2E+05	2	2	2	2	29	0	30	0	0	0.9	0	0.9	0	0	18	6	2	1	2	2	0	1
28	2	2	2	2	2	1	80000	2	2	2	2	46	50	0	35	0	1.1	1.4	0	1.1	0	30	5	2	2	2	2	0	1
29	2	2	2	2	2	1	2E+05	1	2	2	2	30	0	31	0	0	0.5	0	0.7	0	0	15	4	2	2	2	2	0	1
30	1	1	2	2	2	1	90000	2	2	2	2	34	0	29	0	0	0.9	0	1	0	0	25	6	2	2	2	2	0	1
31	2	2	2	2	2	1	1E+05	2	2	2	2	30	0	32	0	0	0.9	0	0.7	0	0	18	5	2	2	2	2	0	1

32	2	2	2	2	2	1	3E+05	1	2	2	2	18	0	0	0	0	0.5	0	0	0	0	10	3	2	2	2	2	0	1
33	2	2	2	2	2	1	1E+05	2	2	2	2	28	0	30	0	0	0.9	0	0.8	0	0	15	5	2	2	2	2	0	1
34	2	2	2	2	2	1	1E+05	2	2	2	2	41	0	0	40	0	1.1	0	0	0.9	0	18	6	2	2	2	2	0	1
35	1	1	1	2	2	1	2E+05	1	2	2	2	27	0	27	0	0	0.8	0	0.9	0	0	15	4	2	2	2	2	0	1
36	2	2	2	2	2	1	2E+05	1	2	2	2	19	0	0	0	24	0.6	0	0	0	0.8	15	8	2	1	2	2	0	1
37	1	1	1	2	2	1	2E+05	1	2	2	2	26	0	0	0	0	0.8	0	0	0	0	20	4	2	2	2	2	0	1
38	1	1	2	2	2	1	1E+05	1	2	2	2	20	0	0	40	0	0.8	0	0	1	0	20	6	2	2	2	2	0	1
39	1	1	2	2	2	1	2E+05	1	2	2	2	20	0	0	0	35	0.6	0	0	0	1	20	8	2	1	2	2	0	1
40	2	2	2	2	2	1	2E+05	2	2	2	2	29	0	0	30	0	0.8	0	0	0.8	0	20	6	2	2	2	2	0	1
41	2	2	2	2	2	1	1E+05	2	2	2	2	26	0	28	0	0	0.7	0	0.8	0	0	10	3	2	2	2	2	0	1
42	2	2	2	2	2	1	1E+05	2	2	2	2	28	0	0	29	0	0.7	0	0	0.6	0	18	4	2	2	2	2	0	1
43	1	1	2	2	2	1	1E+05	2	2	2	2	19	34	0	0	0	0.8	0.9	0	0	0	18	4	2	2	2	2	0	1
44	2	2	2	2	2	1	2E+05	2	2	2	2	23	0	25	0	0	0.7	0	0.9	0	0	23	4	2	2	2	2	0	1
45	1	1	2	2	2	1	3E+05	2	2	2	2	40	0	0	34	35	0.9	0	0	0.8	0.7	10	7	2	2	2	2	0	1
46	1	1	2	2	2	1	2E+05	1	2	2	2	21	0	29	0	0	0.7	0	0.8	0	0	15	4	2	2	2	2	0	1
47	2	2	2	2	2	1	1E+05	2	2	2	2	20	0	0	24	0	0.7	0	0	0.8	0	10	4	2	2	2	2	0	1
48	2	2	2	2	2	1	2E+05	2	2	2	2	24	0	0	0	0	0.8	0	0.8	0	0	15	4	2	2	2	2	0	1
49	2	2	2	2	2	1	1E+05	2	2	2	2	28	0	34	36	29	0.6	0	0.9	1	0.8	24	6	2	2	2	2	0	1
50	2	2	2	2	2	1	2E+05	2	2	2	2	20	0	0	0	26	0.9	0	0	0	0.8	16	7	2	1	2	2	0	1
51	2	2	2	2	2	1	2E+05	2	2	2	2	21	0	28	0	0	0.6	0	0.8	0	0	13	3	2	2	2	2	0	1
52	2	2	2	2	2	1	60000	2	2	2	1	28	0	0	28	0	0.9	0	0	1	0	20	5	2	2	2	2	0	1
53	2	2	2	2	2	1	94000	2	2	2	2	28	0	32	0	0	0.8	0	1	0	0	16	4	2	2	2	2	0	1
54	2	2	2	2	2	1	1E+05	2	2	2	1	31	0	29	0	0	0.9	0	0.9	0	0	25	5	2	2	2	2	0	1
55	2	2	2	2	2	1	2E+05	2	2	2	2	20	0	24	0	0	0.6	0	0.7	0	0	15	4	2	2	2	2	0	1
56	2	2	2	2	2	1	2E+05	2	2	2	2	20	0	24	0	0	0.9	0	0.7	0	0	18	3	2	2	2	2	0	1
57	2	2	2	2	2	1	1E+05	2	2	2	2	18	26	0	0	0	0.5	0.7	0	0	0	15	4	2	2	2	2	0	1
58	1	1	2	2	1	1	2E+05	1	2	2	2	18	40	0	0	35	0.4	1.2	0	0	0.9	23	8	2	2	1	2	0	1
59	1	1	2	2	2	1	3E+05	2	2	2	2	28	0	0	0	0	0.9	0	0	0	0	16	3	2	2	2	2	0	1
60	2	2	2	2	2	1	43000	2	2	2	1	27	0	0	30	30	1	0	0	0.9	0.8	30	8	2	2	2	2	0	1
61	2	2	2	2	2	1	3E+05	2	2	2	1	40	0	33	0	0	1	0	0.8	0	0	18	4	2	2	2	2	0	1
62	2	2	2	2	2	1	2E+05	2	2	2	2	17	0	0	0	0	0.4	0	0	0	0	19	3	2	2	2	2	0	1
63	2	2	2	2	2	1	3E+05	2	2	2	2	26	0	0	0	0	0.7	0	0	0	0	15	3	2	2	2	2	0	1
64	2	2	2	2	2	1	1E+05	2	2	2	2	18	0	0	0	0	0.6	0	0	0	0	10	4	2	2	2	2	0	1
65	2	2	2	2	2	1	2E+05	2	2	2	2	25	34	0	0	0	0.6	1	0	0	0	8	4	2	2	2	2	0	1
66	2	2	2	2	2	1	1E+05	2	2	2	2	24	0	27	0	0	0.8	0	0.7	0	0	20	4	2	2	2	2	0	1
67	1	1	1	1	1	1	3E+05	1	2	2	2	22	21	28	26	28	0.6	0.8	0.9	0.8	0.8	15	7	2	2	1	2	0	1
68	2	2	2	2	2	1	70000	2	2	2	1	28	0	30	0	0	0.6	0	0.8	0	0	18	4	2	2	2	2	0	1
69	1	1	2	2	2	1	1E+05	1	2	2	2	28	0	31	0	0	0.9	0	0.8	0	0	10	3	2	2	2	2	0	1
70	2	2	2	2	2	1	2E+05	2	2	2	2	40	0	30	0	0	1.1	0	0.8	0	0	13	3	2	2	2	2	0	1

71	1	1	2	2	2	1	23000	2	2	2	1	20	29	30	0	28	0.6	0.1	1	0	0.7	30	7	2	2	2	2	0	1
72	2	2	2	2	2	1	2E+05	2	2	2	2	39	37	0	0	0	1.1	1	0	0	0	20	5	2	2	2	2	0	1
73	2	2	2	2	2	1	60000	2	2	2	1	28	40	0	34	0	0.9	1	0	0.8	0	30	6	2	2	2	2	0	1
74	1	1	1	1	2	1	2E+05	1	2	2	2	30	0	31	0	30	0.9	0	0.7	0	0.8	15	5	2	2	1	2	0	1
75	1	1	2	2	2	1	2E+05	1	2	2	2	30	0	0	30	0	0.8	0	0	0.7	0	18	4	2	2	2	2	0	1
76	2	2	2	2	2	1	2E+05	2	2	2	1	38	0	35	0	0	0.9	0	0.6	0	0	20	4	2	2	2	2	0	1
77	2	2	2	2	2	1	2E+05	1	2	2	2	39	0	0	36	0	0.9	0	0	0.8	0	15	7	2	1	2	2	0	1
78	1	1	1	2	1	1	2E+05	1	2	2	2	28	30	29	31	30	0.9	0.8	0.9	0.9	0.8	20	5	2	2	1	2	0	1
79	2	2	2	2	2	1	50000	2	2	2	2	30	40	34	0	28	0.8	0.7	0.7	0	0.8	20	5	2	2	2	2	0	1
80	2	2	2	2	2	2	52000	2	2	2	1	48	54	60	51	40	1	1.3	2.1	1.3	1.1	25	6	2	2	2	2	0	1
81	2	2	2	2	2	2	60000	2	2	2	1	80	86	64	40	34	2.2	2.8	2	1.4	1	25	8	2	2	2	1	3	1
82	2	2	2	2	2	2	74000	2	2	2	1	18	90	88	54	38	0.6	1.8	2	1.2	0.9	20	6	2	2	2	2	0	1
83	2	2	2	2	2	2	1E+05	2	2	2	2	28	78	64	51	38	0.6	2.8	2	1.4	0.8	30	6	2	2	2	2	0	1
84	2	2	2	2	2	2	90000	2	2	2	1	17	70	98	65	34	0.4	2.3	3.2	1.8	0.9	21	8	2	2	2	1	3	1
85	1	1	1	1	1	2	1E+05	2	2	2	2	38	25	97	64	39	0.9	1.6	1.8	1.4	1.2	20	30	2	2	1	2	0	1
86	2	2	2	2	2	1	1E+05	2	2	2	2	33	70	63	49	34	1.1	1.7	1.7	1.4	0.9	15	6	2	2	2	2	0	1
87	2	2	2	2	2	2	1E+05	2	2	2	1	33	45	0	43	36	1.1	1.9	0	1.2	0.7	25	6	2	2	2	2	0	1
88	2	2	2	1	2	2	2E+05	2	2	2	1	65	70	60	54	39	1.8	2.1	1.4	1.2	0.8	21	6	2	2	1	2	0	1
89	1	1	1	1	2	2	1E+05	2	2	2	1	41	70	45	42	34	1.1	1.6	1.3	1	0.5	25	11	2	1	1	2	0	1
90	2	2	2	2	2	2	1E+05	2	2	2	1	56	73	40	0	38	1.3	1.7	1	0	1	15	7	2	2	2	2	0	1
91	2	2	2	2	2	2	2E+05	2	2	2	2	61	62	0	40	39	2	1.8	0	1	0.9	18	5	2	2	2	2	0	1
92	2	2	2	2	2	2	1E+05	2	2	2	2	36	77	62	48	35	0.7	1.9	2.4	1.6	0.9	20	7	2	2	2	2	0	1
93	2	2	2	2	2	2	1E+05	2	2	2	1	42	96	80	67	50	0.9	3.2	2	1.4	0.9	23	8	2	2	2	1	3	1
94	2	2	2	2	2	2	68000	2	2	2	1	90	104	92	85	69	1.5	4	3.1	2.2	1.8	28	18	2	2	2	1	2	1
95	2	2	2	2	2	2	90000	2	2	2	1	44	102	84	52	38	1.3	3	2.1	1.3	0.9	30	9	2	2	2	2	0	1
96	2	2	2	2	2	2	2E+05	2	2	2	1	32	59	64	51	35	1.6	1.7	2.1	1.3	0.9	24	7	2	2	2	2	0	1
97	2	2	2	2	2	2	90000	2	1	1	1	55	90	0	0	0	1.8	2.3	0	0	0	25	2	2	2	2	2	0	3
98	1	1	1	1	1	2	3E+05	2	2	2	1	53	110	143	0	0	1.6	2.8	2.9	0	0	26	2	2	2	2	2	0	3
99	1	1	2	2	2	1	2E+05	1	2	2	2	33	0	0	35	0	1.1	0	0	1	0	21	4	2	2	2	2	0	1
100	2	2	2	2	2	2	98000	2	2	2	1	73	86	80	64	31	1.3	1.6	1.6	1.4	1.1	25	7	2	2	2	2	0	1

KEY TO MASTER CHART

ES	-	Snake seen
FMS	-	Fang Mark Seen
SOB	-	Site of bite
RR	-	Respiratory Rate
H1	-	Bleeding from bite site
H2	-	Mucosal bleed
H3	-	Epistaxis
H4	-	Petechiae / Ecchymoses
H5	-	Hematuria
H6	-	Hematemesis
H7	-	Hemoptysis
N1	-	Ptosis
N2	-	Opthalamoplegia
N3	-	Pharyngeal Paralysis
N4	-	Respiratory failure
N5	-	Motor Paralysis
WBCT	-	Whole Blood Clotting Time
U1	-	Albumin
U2	-	Sugar
U3	-	RBC
UR 1- UR5	-	Urea (Day1-Day 5)
CR1 - CR5	-	Creatinine (Day1-Day5)
ASV	-	Anti Snake Venom

பங்கேற்பவர்களுக்கு ஆய்வின் விவரம்

ஆய்வின் நோக்கம்:

பாம்பு கடியினால் பாதிக்கப்பட்ட உள்நோயாளிகளின் மருத்துவ சுயவிவரம் மற்றும் அதனால் ஏற்படும் தீவிர சிறுநீரக செயலிழப்பு பற்றிய ஆய்வு செய்தல்.

ஆய்வில் பங்கேற்க தகுதிகள்:

1. பாம்பு விஷக்கடி நோயாளியால் உறுதி செய்யப்படவேண்டும்.
2. ஏற்கனவே சிறுநீரக பாதிப்பு உட்படாதவறாக இருக்க வேண்டும்.

செய்முறை விளக்கம்:

இந்த ஆய்வில் பங்கேற்கும் நோயாளிகளிடம் இருந்து மருத்துவ சுயவிவரம் மற்றும் சிறுநீரக செயலிழப்பின் அறிகுறிகள் குறித்து விவரங்கள் சேகரிக்கப்படும்.

மருத்துவமனையில் உள்நோயாளியாக அனுமதிக்கப்படுவர்களுக்கு செய்யப்படும் வழக்கமான ரத்த பரிசோதனை, சிறுநீர் பரிசோதனை ECG குறித்த விவரங்கள் நோயாளிகளிடம் இருந்து பெறப்படும்.

சுய ஒப்புதல் படிவம்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொண்டேன். நான் ஆய்வில் இருந்து விலகி கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்தி கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் .....

நாள் .....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....

நாள் ..... ஆய்வாளரின் பெயர் .....